The Journal of Occupational Diseases and Traumatic Surgery

Industrial Medicine

for

Physicians and Surgeons in Industry

Volume 6 JANUARY, 1937 Number 1

THIS ISSUE

Arthritis
—Personal Views and Some Recent Developments—

The Curability of Syphilis
—Importance of the Time Element—

Resuscitation
—of the Apparently Dead from Electric Shock—

Cancer Development
—from Chronic Irritation—

Abdominal Trauma
—and Its Diagnosis—

Pre-Employment Examinations
—A Digest of the First Thousand of a Series—

Carbon Tetrachloride Poisoning
—Outline of a Successful Treatment—

Hysteria and Malingering
—The Differential Diagnosis from Traumatic Organic Disease of the Central Nervous System—

Pathology of the Pneumoconioses
—Silicosis; Silicosis and Tuberculosis; Asbestosis; Siderosis; Anthracosis—

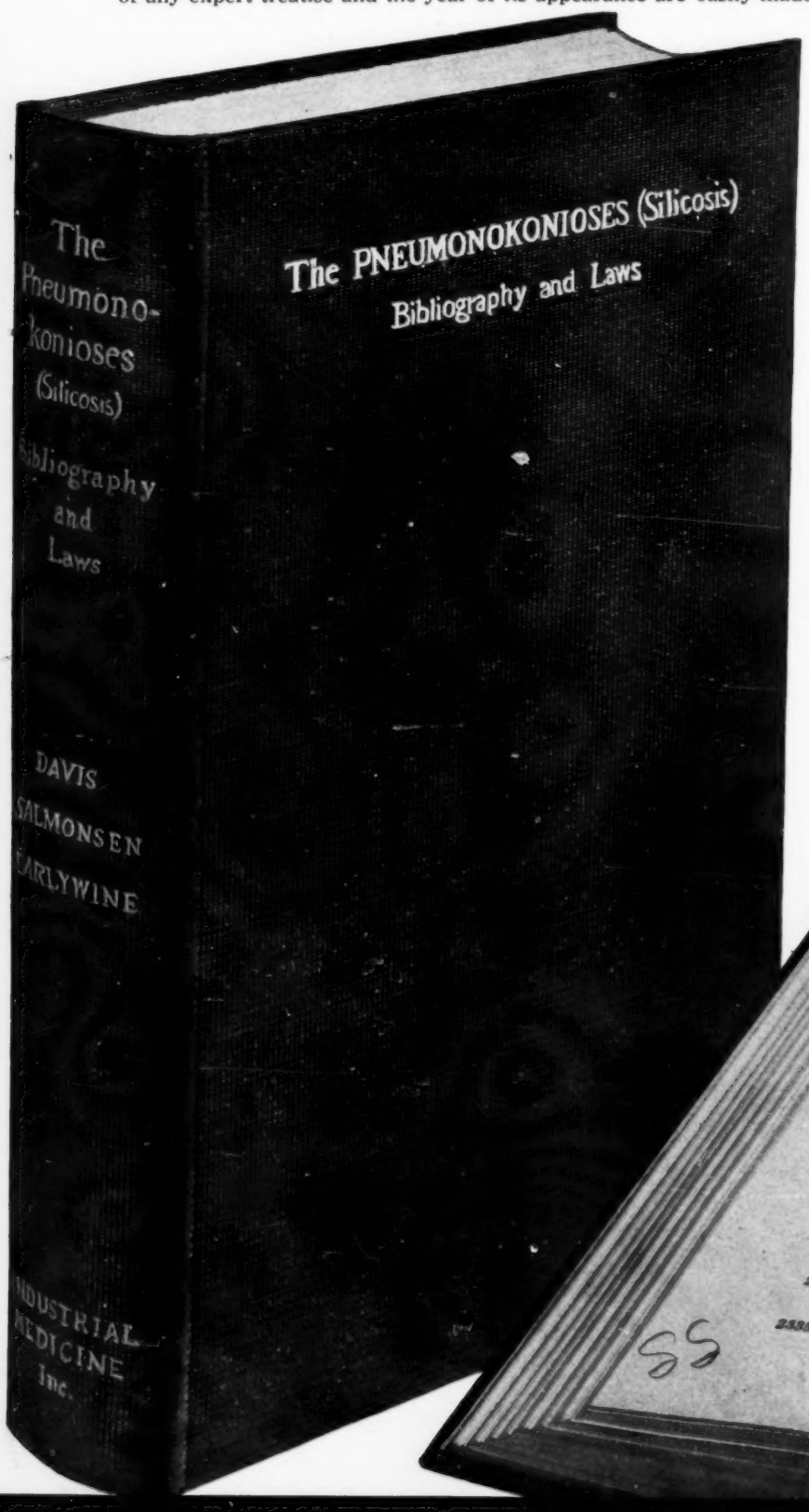
The Pneumonokonioses (Silicosis)

R v.6 Jan.-June 1937

Bibliography *and* Laws Book No. I

All the References of the World's Literature
--- from 1556 to December 31, 1933 ---

Review from **"Staub"**, (Dust), 1, No. 1:167-168, April, 1936, done by Dr. Ing. O. M. Faber, Berlin.
No. 115. Bibliography and Laws upon Silicosis. **(The Pneumonoconioses (Silicosis) Bibliography and Laws)**, by Geo. G. Davis, E. M. Salmonsen and J. L. Earlywine, published by **Industrial Medicine**, Chicago, Ill., 1934, 482 pp. ($7.50).
The **Forword** is by E. R. Le Count, Chief of the Department of Pathology, The University of Chicago. The book has two main divisions. Part I is a Bibliography of the collected literature of the world upon the **Pneumonoconioses (Silicosis)**. Beginning with the **De Re Metallica** of Agricola (1556), it ends with 1933, inclusive of 2,768 sources, whose title, author and exact citation are given. The works are chronologically arranged. Through subdivision into different groups, the works of authors appearing in the same year are clearly and immediately perceived. Within the groups the works are still arranged in alphabetical sequence by authors. These 276 pages of comprehensive bibliography are followed by a complete **author—and subject—index.** Besides, the location (orientation) of any expert treatise and the year of its appearance are easily made out at a glance.



In Part II, the Laws of the several States of the Union in North America, as relating to Occupational Diseases, the injuries they cause, and any pertinent material, are all represented. The plan and sub-divisions by which the situation can be ascertained for any one of the 48 states of the United States of America, are readily apprehended. In several cases corresponding paragraphs have cross-references. Especially valuable and complete is this legal survey for ascertaining any characteristic verdict (judgment) in respect of a pneumonoconiosis, showing the basis of the action, the conditions giving rise to the action, as well as the principle involved in the decision—for any state in the Union. The book closes with an alphabetical summary of the enterprises concerned in the juridical distinctions (or decisions.)

Examination of the book compels recognition of the extraordinary care and pains taken by the compilers. Random tests here and there show that for a space of 377 years this bibliography has attained a completeness not hitherto arrived at. Probably no branch of any other science has at its disposal so flawless and richly extensive a bibliography. It is authoritative not only for physicians and industrial experts, but also for physicists, engineers, and employers of industry. The format is entirely appropriate; for the arduous work involved the compilers should receive the fullest recognition.

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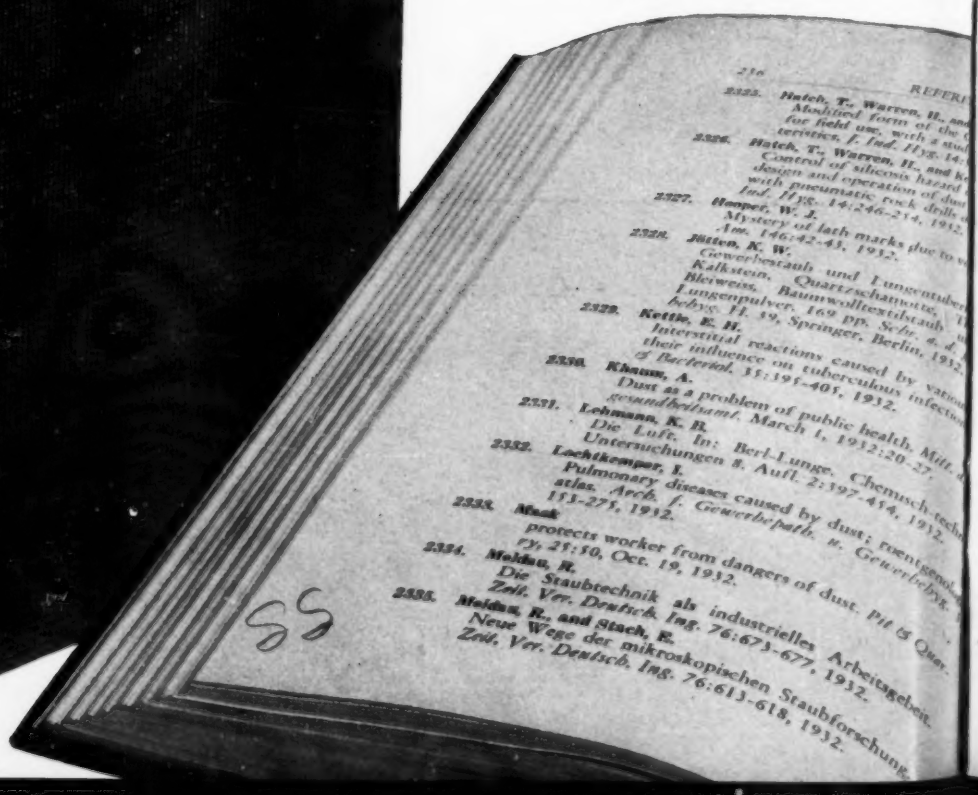

The

Pneumonokonioses (Silicosis) Book No. II

—Literature and Laws of 1934—

International Abstracts, Extracts and Reviews of the 1934 Literature of the Pneumonokonioses and Their Allied Diseases and Subjects—Plus the 1934 Bibliography.

Compiled by

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The Wisconsin Medical Journal

BOOK REVIEW

The Pneumonoconioses (Silicosis). Literature and Laws of 1934. By George G. Davis, M.D., Associate Clinical professor of surgery, Rush Medical College, University of Chicago, attending surgeon Cook County Hospital.

A year ago Dr. Davis and his collaborators published Book I which listed, with few exceptions, all the references to pneumonokonioses from 1556 to 1933 inclusive.

The present volume (Book II) furnishes a review of the literature and laws of 1934. Whereas Book I merely cited references, the present work furnishes an abstract of each article. The work is complete. There is a total of 396 references. In addition, the bibliography, not included in Book I, is included. There are good subject and author indexes.

This volume together with Book I will be very valuable to workers in this field. The extensive literature of but one year is indicative of the wide and increasing interest that this subject holds. Compilation of this book has been a worthwhile project. O. O. M.—*Issue November*, 1935.

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OCCUPATIONAL DISEASES
TRAUMATIC, INDUSTRIAL

INDUSTRIAL MEDICINE
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DR. WILLIS C. TEMPLER, who has joined our Board of Editors, is President of the New York State Society of Industrial Medicine, a member of the Conference Board of Physicians in Industry, and, since 1923, Medical Director of Corning Glass Works . . . DR. POST'S distinction as Consultant in Internal Medicine, and, in the words of DR. SLOAN, "the confidence of his close association with the problem over so many years," invest his "Personal Views" on arthritis with timely interest and permanent importance . . . DR. ELLER, author of more than a hundred articles on dermatological and related subjects, and a 700-page "Treatise on Benign and Malignant Tumors of the Skin," (Lea & Febiger, this year) has two very special interests: one the American Dermatological Association; the other the Pan American Medical Association, of which he is the Director General . . . DR. STONE, whose subject is "Hysteria and Malingering," is Assistant Professor of Nervous and Mental Diseases, Northwestern University Medical School . . . DR. BRENNECKE'S connection with railroad surgery dates back to 1906; he is also Chief Surgeon for Western United Gas & Electric and affiliated companies, and Surgeon for numerous manufacturers. Avocationally he is studying pernicious anemia . . . DR. HART FISHER, discussing DR. BRENNECKE'S subject, says "there is no improvement today in the treatment of electrification cases." He speaks with the authority of 20 years' experience . . . DR. BAINBRIDGE'S book, "The Cancer Problem," has been translated into French, Italian, Spanish, Polish, and Arabic editions . . . DR. JAMES T. CASE, in his short review of Cole's Textbook of Surgery, (p. 33), uses the word "crispness" in relation to the author's style. That term also is applicable to the style in which DR. HANCHETT sets forth, in the ordered precision of well-marshalled preparation, such a complete study of "Abdominal Trauma and Its Diagnosis" as will move the reader to concur with DR. HELLER in his statement that "DR. HANCHETT has left relatively little to be said in the matter" . . . DRS. BARSKY

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and WOLFF develop a number of interesting things in their digest of the first thousand of a series of pre-employment examinations. Not the least interesting is expressed in the statement that "the average time per examination was 45 to 60 minutes" . . . DR. DAVIS' "Outline of a Successful Treatment" for a case of carbon tetrachloride poisoning reminds us of a certain chief executive who, in delegating a responsibility, never fails to express his confidence that it will be discharged "in your usual capable and efficient manner." The association of ideas can be short-circuited by the remark of one of our readers: "Any article by GEO. DAVIS is good" . . . DR. AUERBACH'S exhaustive and well-illustrated "Pathology of the Pneumoconioses" was published in the recent Occupational Disease Number of the Quarterly Bulletin of Sea View Hospital, and is given here the wider availability to readers which it deserves. A popular magazine's summary of striking incidents of 1936 has the sentence: "Many heard about silicosis; some knew what it meant." There are those—as the accuser says when he doesn't want to name any names—who would make the sentence more complete by adding: "and a few physicians were able to recognize it when they saw it" . . . THOMAS C. ANGERSTEIN, of the Chicago Bar, is author of the 1200-page textbook: "The Employer and the Workmen's Compensation Act of Illinois." His able article in this issue on "Occupational Diseases and the Illinois Law" has its second paragraph deleted. This is it: "I also take this opportunity to say that INDUSTRIAL MEDICINE, The Journal of Occupational Diseases and Traumatic Surgery, is doing a great work and rendering a very important service in this field, and that it should be used by everyone having to do with occupational diseases, as a means of making available and disseminating information and knowledge, which, as we shall later see, is of fundamental importance in the application and administration of the occupational diseases law, and in the solution of occupational disease problems. So far as possible, back numbers of this excellent publication should also be secured." We took this out from under its heading for two reasons: First, to indicate that we are still sensitive to the differential diagnosis between the kind of pride in achievement which inspires to more and better work and the kind which is the O. D. known as "swell head." And second, to mention that our supply of back numbers has already been drawn upon heavily as to certain issues—and to inquire if anyone wants a bound volume of 1936 numbers.

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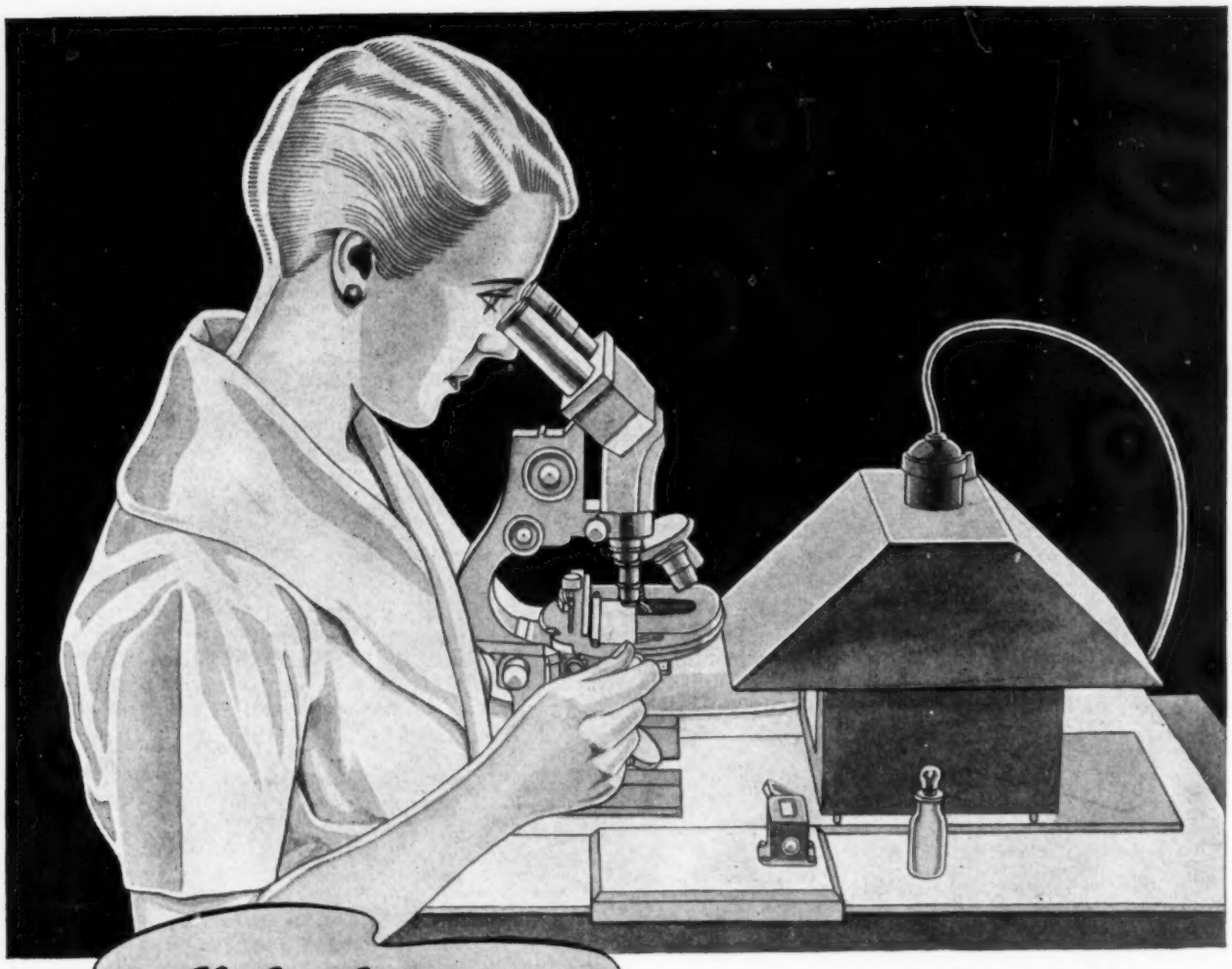

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Industrial Medicine

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The editors will exercise care in checking on the accuracy of data printed, but in all other respects articles and opinions of which expression is allowed are the opinions of their authors—the editors reserving in all cases the right to comment on the same, in the current or any subsequent issues, as they may be inclined.

The Journal of Occupational Diseases and Traumatic Surgery

The Science, the Law and the Economics of Industrial Health

Volume 6 JANUARY, 1937 Number 1

Arthritis

—Personal Views and Some Recent Developments—

WILBER E. POST, M.D.,
Chicago, Illinois

I HAVE no cure-all for rheumatic arthritis; I shun faddism.* I endeavor to study all pertinent phases of pathology in each case of rheumatism. *Thus far* I trust we meet on common ground. Next, I wish to point out that I know that the question of focal infections and rheumatism, i.e., arthritis and neuritis and myositis, is a very old subject. In evidence I refer you to an ancient tablet which records that a physician of Assyria, Arad Nana, wrote of the Prince Ashur-bani-apal these words: "The pain in his head, his sides and his feet has come from his teeth; they must be extracted." The date of this record is thought to be 680 B.C.

Some may question the significance of the title of the discussion as it relates to "Personal Views." I hasten to explain that I do *not* lay claim to any private, heretofore unpublished, communication from the bureau of rheumatism in the world of pathology which gives the causes and processes and classifications of, and remedies for, rheumatism in simple clear fashion and which has been heretofore unknown to the many workers in this important field of human disease. The fact is that when the Chief Surgeon of a railroad called me up and asked what I would discuss at the meeting of the American Association of Railway Surgeons I knew it was a command, began my answer immediately with the words "Personal Views," and then thought of some rather interesting fairly recent developments in our work in arthritis. At any rate we are doubtless agreed that we do not know everything about arthritis or rheumatism, and that there are differences of opinions and experiences among us. So long as that is true there should be discussions on the subject. Certain it is that any disease "which is the greatest single cause of disability in the temperate climates and which produces more pensionable invalidism than any other condition except cardiovascular disease in old age" may well be discussed by American railway surgeons.

It was my privilege to do the bacteriological studies on the first pair of tonsils and the joint tissue removed under Dr. Frank Billings' direction in a case of arthritis when Dr. Billings began his work in what came to be called focal infections, about 1909. That work was soon turned over to Dr. D. J. Davis, now Dean of the University of Illinois College of Medicine. He found streptococci, mostly of the hemolytic strains, in all but two of the first 150 pairs of tonsils removed from arthritic patients in that study. These organisms were found in the *bottom* of the tonsillar crypts. By intravenous injections of small doses of these organisms he was the first to produce experimentally in animals (rabbits) a non-suppurative arthritis which became chronic, involving periarticular and articular tissues and resembling chronic rheumatic arthritis. In 1910 Davis repeated the experiment in Aschoff's laboratory in Freiburg. Later similar studies were carried out on infections about the teeth. Rosenow took up the work here and later has continued the work at the Mayo's. Bacterial vaccines were being used in various forms of infection at that time, and in this work autogenous vaccines were employed over a period of several years. These were prepared from cultures obtained from foci of infection, from periarticular tissues and from lymph nodes regional to affected joints. Modifications of vaccines eliminating the toxic factor and preserving the immunizing portion and various dosages were studied with control by the opsonic index and otherwise. Horses were immunized to many strains of streptococci which had been obtained from arthritics and which had produced arthritis experimentally in animals. The foreign protein of horse serum proved dangerous, of course. The effect of vaccines was often doubtful, or at least uncertain, although apparently beneficial in a small percentage of cases, especially in the subacute infectious types. However, the routine use of even autogenous vaccines was largely abandoned for several years.

IN THE meantime the management of arthritis included removal of demonstrable foci of infection, a nutritious high vitamin diet, a high dry, sunny climate when possible, and heliotherapy, physiotherapy and hydrotherapy with varying emphasis.

Then the studies of Pemberton demonstrated the importance of the metabolic factor in regard to carbohydrates; Graham and Fletcher emphasized intestinal hygiene; then the role of vita-

* Presented at the Forty-Seventh Annual Convention of the American Association of Railway Surgeons, Chicago, November 5, 1936.

mines, Hench's studies in gout, sympathectomy by Adson and others, allergy, endocrine disturbances, especially thyroid dyscrasia and menopausal disturbances of metabolism, and trauma and occupation, senescence and heredity in relation to arthritis, were all discussed anew.

When we summed them up it seemed to me that these various factors in arthritis might well be grouped as follows:

1. Infections.
2. Metabolic disturbances.
3. Trauma.

These main divisions are amplified in Chart I.

I. INFECTIONS
- a. Gonococcus
- b. Haverhillia Multiformis
- c. Meningococcus
- d. Pneumococcus
- e. Staphylococcus (septic)
- f. Streptococcus
  - Pyogenic (septic)
  - Rheumatic
- g. Syphilis
- h. Tuberculosis
- i. Typhoid
- j. Undulant fever

II. METABOLIC DISTURBANCES
- a. Allergy
- b. Chilling
- c. Circulatory disturbances
  - Diminished capillaries
  - Slow blood flow
  - Arteriosclerosis and hypertension
  - Varicosities
  - Hyperemia
  - Anoxemia—Cardiac deficiency
    —Pulmonic fibrosis
- d. Endocrine disturbance
  - Thyroid—obesity
  - Ovary—menopause

  Hypometabolism
  - Pituitary
- e. Fatigue
  - Vasomotor disturbance
    (perhaps adrenal deficiency)
  - Predisposition to infection
- f. Nutritional disturbances
  - Food deficiency or overeating
  - Vitamine deficiency
  - Gastro-intestinal disturbances
  - Disturbance of sulphur and calcium chemistry

III. TRAUMA
- a. Microtraumata
  - Obesity
  - Occupation
  - Posture
- b. Injuries

Chart I. Etiologic Factors in Arthritis

This has been my routine working classification. That is, it is the basis of inventory of the various factors in each case of arthritis which seeks my advice. We keep in mind also that more than one factor deserves attention in most cases. This in no way violates the spirit of the classification recommended by the American Committee for the Control of Rheumatism (See Chart II.) Rather, it conforms to it.

So far as "rheumatic" disease is concerned, we have:

Rheumatic Fever.

Atrophic Arthritis (Chronic infectious, rheumatoid arthritis, proliferative, synovial.)

Hypertrophic Arthritis (Osteoarthritis. chondro-osseous arthritis, chronic senescent, degenerative.)

Fibrositis (Rheumatic infection of muscles and fibrous tissues.)

Some may question the inclusion of chronic hypertrophic arthritis in the group of rheumatic joint diseases. We do include it because infection is still considered by many as being *one* of the causative factors although the pathologic changes are chiefly degeneration with bone hypertrophy, increased cholesterol in the blood plasma and less frequent agglutinin and antihemolysin reactions.

The management of arthritis, as based upon our knowledge of the causative factors and the manifestations, came to include:

1. Removal of sources of infection with improvement of thoroughness and judgment.
2. Vaccine or foreign protein therapy, probably in fewer cases and with better selection of cases, and much less often as a cure-all.
3. Physiotherapy, hydrotherapy, diathermy, fever therapy, heliotherapy, climatotherapy as appropriate and available.
4. Diet managed with variations according to one's views regarding carbohydrates and fats and high and low calories but usually including vitamine-rich foods.
5. Gastro-intestinal hygiene.
6. Medicines, including salicylates, arsenic, iodides, iron, liver extract, cod liver oil, yeast, viosterol, calcium ortho-iodoxy benzoate, gold and sulphur preparations.
7. Surgery, including orthopedic measures removal of fluid from joints and bursae, removal of loose cartilages, resection of synovial membranes.

AMONG the helpful developments in our knowledge and methods in rheumatic joint disease in the last several years has been (1) the use of the toxin of the streptococcus in the filtrate of its bouillon culture diagnostically and therapeutically, and (2) the measurement of the cataphoretic potential (i.e., the negative electric charge) of the streptococcus as a means of identifying the arthrotropic strains of the organism, and helping to determine foci of infection, the reaction of patient's serum and thus the identification of the type of arthritis in a patient.

I. Those attributable to trauma.
II. Those attributable to infections of known type.
III. Those possibly or probably attributable to infection or related toxins, for example:
  - Rheumatic Fever,
  - Atrophic Arthritis (Chronic infections, rheumatoid, proliferative)

IV. Those of which the chief characteristic is Degenerative change in tissue, for example:
  - Hypertrophic Arthritis (senescent, degenerative, osteoarthritis)

V. Those of which the chief or only obvious characteristic is some recognizable or suspected chemical derangement—chemical, metabolic, allergic, endocrine.
VI. Miscellaneous group of unclassified types.

Chart II. Scheme of Classification of Diseases of Joints. Published by the American Committee for the Control of Rheumatism

This development came to me in this way. In 1923 and 1924 the Doctors Dick announced their success in making an effective scarlet fever antitoxin by the use of toxin of scarlet fever streptococcus as antigen. This toxin was obtained in the filtrate of the bouillon culture. It occurred to me that we might logically use the same principle in the study and treatment of rheumatic disease inasmuch as a streptococcus infection is involved.

Accordingly, Dr. Willard L. Wood and I have been pursuing such studies for six years.

When the filtrate of an autogenous broth culture containing this toxin is injected in small doses into the skin of the patient, three things may occur:

1. The local reaction may be very marked causing a large area of redness, swelling, burning and soreness lasting 24 to 48 hours;
2. Previously affected joints and bursae may become very painful for a period of a few hours to one week;
3. The tissues at the site of the original focus of infection, such as ethmoids or sinuses or throat, may become swollen and painful.

The reaction resembles that seen in an old tubercular lesion when old tuberculin is injected subcutaneously. Similar reactions in joints had been observed previously from time to time in cases of rheumatic arthritis when autogenous vaccines were injected subcutaneously. These vaccines were essentially suspensions of dead bacteria, while the material used in our recent work is a filtrate of the broth culture of the organism. I do not remember a case in which the tissues at the site of the original focus were disturbed by injections of vaccines, while such disturbances have been rather frequent and sometimes marked since the filtrate of broth culture has been used. One of the benefits of the latter is that previously obscure foci of infection may be revealed so that appropriate steps may be taken to eradicate the infection. Further, the affected tissues about joints and in bursae may improve, sometimes remarkably, following the reaction. In one case I have seen even the calcareous deposit of long standing in a subdeltoid bursa entirely disappear (possibly due to the hyperemia induced). Indeed, this is the basis for the use of the toxic bouillon filtrate as a therapeutic agent. Such reactions are also an indication that the toxin used is specific for that patient's infection.

The determination of the cataphoretic potential of bacteria (i.e., the measurement of the negative electric charge of bacteria) and its application in the field of focal infection and other infectious diseases has seemed to me almost a fairy tale or turn of magic. The method consists of observing through a microscope the rate of speed at which bacteria in suspension will move from anode to cathode under a measured voltage. The method has been known to physics for some years and used to determine the electric charge of particles in suspension. Within recent years the procedure has been used in bacteriology. Lloyd B. Jensen, working with Falk at the University of Chicago, applied the method in differentiating various strains of diphtheria bacilli. He found that the same strains of bacilli cultured in definite and identical conditions always moved in suspension from anode to cathode at the same rate of speed. Another important observation was that nonpathogenic organisms or organisms once pathogenic which have lost their pathogenicity have no electric charge, and, therefore, do not move from anode to cathode in such suspensions.

It occurred to Jensen that the method might be applied in the differentiation of various strains of streptococci. This was suggested to Dr. E. C. Rosenow, and Dr. Jensen was promptly invited to Rochester. The result was that in tests of 5,000 strains of streptococci the grouping according to their cataphoretic potential corresponded to the grouping already made by Rosenow by cultural, biologic and clinical characteristics.

Further, when streptococci of arthrotropic potential are suspended in dilutions of serum of patients suffering from streptococcus (rheumatic) arthritis, or when streptococci of neurotropic potential are suspended in dilutions of serum of patients suffering from streptococcus neuritis, the streptococci in suspension will not move from anode to cathode. Something in such a patient's serum causes the bacteria to lose their negative electric charge. We suppose an antibody in the serum covers or combines with the bacteria and neutralizes or overcomes its negative charge. The sera of different patients differ in their power to decrease the potential of these bacteria, and the serum of the same patient may vary with time, probably according to his immunity.

1. Rheumatoid arthritis: Cases studied — 175
2. Neuritis (infectious): Cases studied — 95
3. Cases with definite improvements:
   (Alleviation of symptoms)
   a. Arthritis — 145 or 83%
   b. Neuritis — 85 or 90%

   Total — 230 cases

	No. of Cases
Medical treatment only	35
Removal of foci only	45
Removal of foci plus filtrate	80
Filtrate only	70
Total	230

4. Kinds of organisms isolated, studied, and used in filtrates:
   a. Strep vir in about 75% of the cases.
   b. Strep hem in about 20% of the cases.
   c. Hem staph albus and aureus and indifferent strep in about 5% of the cases.

Chart III. Cases

Thus we have another method of determining electrically whether streptococci obtained from the nose or throat or teeth or prostate may be the cause of arthritis or neuritis, and also, a method of determining whether the serum of a patient suffering from an arthritis or neuritis contains an antibody for the arthrotropic or neurotropic streptococcus, respectively.

Let me emphasize that our studies even in cases of rheumatoid (infectious) arthritis (and of infectious neuritis) included a careful search for all possible known factors other than infection, and that appropriate attention was given to these factors. Removal of foci was done when the focus was demonstrable and when it was possible to remove the focus. In addition, increasing doses of the autogenous toxic filtrate were injected at five-day to one-week intervals up to 20 or 30 doses. At the same time in some cases the toxic filtrate was sprayed into the nose and throat by a nebulizer in an effort to stimulate the protective mechanism of those tissues.

NOW a word about results. Exact figures are not compiled, but our experience shows much better results since these methods were used. Of cases of rheumatoid arthritis 175 have been thus studied and treated, and 145 (or 83%) have shown definite improvement. Of cases of infectious neuritis 95 have been studied and 85 (or 90%) have shown definite improvement. From

this point the figures at hand include both the arthritis and neuritis cases—230 in all; the figures are approximate but, I believe, reliable. Medical treatment alone was used in 35, removal of foci alone in 45, removal of foci and injections of toxic filtrate were used in 80, injections of toxic filtrates alone in 70. (See Chart III).

One of the strangest impressions received in our experience with these methods is the improvement and some apparent cures in cases who have resisted other methods. The term "cure" is not used because recurrences may develop in what appear now to be cures.

Discussion:

LE ROY H. SLOAN, M.D., F.A.C.P., Chicago: I shall not try to add to the material presented by Dr. Post in his very excellent paper. It has been a great satisfaction to hear Dr. Post, largely because we have the confidence of his close association with the problem of arthritis over so many years.

To many of us it has been a source of discouragement not to be able to isolate organisms from the bloodstream of patients who, we felt, had the bacteriemia of rheumatism. The whole clinical picture of rheumatism, both the acute rheumatic fever and the subacute rheumatic conditions, has pointed to a bloodstream infection, but how rarely have we been able to isolate from such patients definite organisms which we felt were really of etiological significance. Even more disturbing in the last few years is to have one worker report 90% of positive blood cultures on acute rheumatic fever and on chronic arthritis even of the type which we ordinarily call atrophic arthritis, while other workers using at times the same material have been able only to find perhaps 10% positive blood cultures.

This work of Dr. Post comes to us, therefore, with a greater significance. We have all felt that arthritis in its usual picture was of infectious origin, yet we could not prove it. Dr. Post's work, with that of his associates, points the way and gives a hope for this proof.

In the past we have been quite well acquainted with infections in the teeth, tonsils, sinuses, but we not infrequently forget to give due attention to the prostate and seminal vesicles, and also we must give more attention to posterior urethral glands and to bowel conditions.

Our great desire has been first to find an organism; then to determine whether this organism might be related to the condition at hand; then to classify this organism, and finally to determine whether the vaccine made from the organism or the filtrate was of real service in the treatment of the condition. This sequence has been worked out for us by Dr. Post, Dr. Wood, and their associates.

We cannot object to the classification presented by Dr. Post. Of course, we know that more than one factor may be at work, as in the very obese patient with a focal infection who continues to traumatize her knees by her overweight and who at the same time may have a marked metabolic lack as the cause of her obesity.

One must not forget our newly found friend, resurreced from the not too distant past, namely, gout. This condition seems to be increasing in frequency.

The treatment of the patient with arthritis becomes, therefore, a treatment of the whole patient, an individualization of treatment, a consideration of his diet, his metabolism, his focal infection, his blood condition, his work, his habits, etc.

The Curability of Syphilis

—Importance of the Time Element—

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New York City

THERE are at least three interpretations of the term "cure" as related to syphilis. The pathologist, the public health official and the clinician may each regard the curability of this disease from different points of view. Affirmation or denial of the possibility of cure must in each case be qualified, according to whether one has in mind radical cure or symptomatic cure. Serologic cure has little significance and does not enter into the discussion, since it is known that anatomic and pathologic changes take place frequently even in the presence of sero-negativity and normal spinal fluids.

The dividing line between curable and incurable syphilis, in the strict or radical sense, is found to be well towards the beginning of the disease process. Any degree of confidence in the curability of syphilis demands that physicians think in terms of hours or days in the duration of the infection rather than in terms of weeks or months. Fortunately, the same concept coincides with that related to the control of infectiousness which is the *sine qua non* of prevention, hence is also the very crux of the question of curability.

When applied to syphilis, the word "cure" must be modified and defined, since it is obvious that such a designation would be improper if applied to the entire gamut of pathology associated with the disease. Indeed, from the syphilo-therapeutic standpoint, its variegated pathologic manifestations scarcely allow us to regard it as one and the same disease. As many syphilologists realize, the curability of syphilis is yet today a clinical presumption instead of an established fact. Our therapeutic optimism allows us to speak of little more than "symptomatic cure" or symptomatic arrest in the late neural, visceral and cutaneous localizations. Cure in this sense distinctly falls short of conveying a true concept; if we can arrest the progress of late syphilis or relieve the symptoms attendant upon an aortitis or a posterior nerve root involvement, then curability must be construed with its true meaning. Even for the latent phase, before the occurrence of any clinical detectable anatomo-pathologic changes, it is extremely doubtful that the disease can ever be biologically exterminated, unless it be along the lines of fever therapy which has given us some encouragement in the past few years.

Curability in the strict or biologic sense will probably always lie well beyond the domain of long standing syphilitic infection. "The golden opportunity," as pointed out by Pusey some years ago, comes but once in the entire course of the disease. It is my belief that physicians who seize upon this moment can radically and completely cure their patients in the great majority of instances, it is my opinion that this expresses the general sentiment of syphilologists today. The few exceptions will depend upon incidental modifying factors as for example, individual drug-intolerance, or possibly arsenic-resistant strains of treponema pallidum. Therefore, the physician can and should promise radical extinction of the infection in the patient whose cooperation will allow the full force of our present-day knowledge of syphilo-therapeusis to be brought to bear upon his case.

Confirmation of the presumptive radical curability of early syphilis must yet depend upon statistical study and investigation for some years to come. At the present time, the most significant facts are perhaps revealed by the U. S. Public Health Service report cooperating with five important American clinics. It has been shown that when a continuous system of treatment (as compared with an "intensive," "intermittent" or "irregular" system) is carried out, sero-negative

primary syphilis gives 85-86% best results. This implies the clinical belief that radical cure is obtainable in an equal percentage of cases. The proportion of such results in sero-positive cases fell to 64-70% and in the secondary phase (with positive blood serology) the cures in the presumptive biological sense amounted to about 60-80%. The statistics pertaining to early syphilis are drawn from about 3,000 cases. Such studies as these, supported by personal observation and follow-up over an appreciable number of years, suffice to indicate my belief that the absolute curability of early syphilis is possible in the majority of individual cases. From the public health point of view, it must not be forgotten that a certain percentage of clinical and serologic relapse should be subtracted from the brief figures mentioned.

These comments serve to emphasize two fundamental requisites, necessary even though insufficient for the radical cure of syphilis. With the earliest possible diagnosis, which is the first essential, and with the immediate institution of treatment according to the best modern standards of a continuous regime, the chances of permanently ridding the body of the infection are excellent, but become progressively less with the lapse of time. Every cutaneous or mucosal lesion entering within the possibility of a primary luetic lesion should be repeatedly subjected to dark-field examination until syphilis has been definitely excluded. Likewise, the diagnosis should be unquestionably established by dark-field examination or confirmed by repeated positive serology before the institution of any treatment whatever. Short prophylactic courses of arsenicals or heavy metals must be unequivocally condemned. If treatment is begun, the entire course should be pursued up to the 18 months. Nothing invites muco-cutaneous, serologic, relapse or late visceral complications more than the inadequate and poorly spaced therapy of early syphilis. Such procedures, totally out of keeping with our actual knowledge of the management of syphilis, sweep forever beyond the physician's grasp the possibility of cure in the individual case.

The factors upon which total extinction of the early infection depends are multiple. Ehrlich's dream of sterilization by a single dose of arsphenamine (Therapia Sterilizans Magna) has fallen short of complete realization, but his work marked the dawn of a new era in the understanding and management of the disease. The trials and errors of the last two decades have now resulted in a very tangible control of curability and infectiousness. The progress has, in fact, been so apparent that it is to be hoped another centennium will find syphilis genuinely rare.

The management of early syphilis resolves itself today into standard treatment procedures, the chief advantages of which lie in a continuous program without rest intervals, combining the arsenicals and heavy metals. The incidence of muco-cutaneous and serologic relapse and the questions of individual drug idiosyncrasies, as well as the possibility of arsenic-resistant strains or ultramicroscopic forms of treponema, preclude a blanket assertion of the positive curability of syphilis. While there are no absolute criteria of cure in the individual case, the physician has every right to promise lifelong freedom from any signs or symptoms of the disease and the impossibility of transmission either to other individuals or to offspring. As with all transmissible diseases, the keynote of curability is struck in the prevention of infectiousness. Rendering the individual case non-infectious is indeed the most important aim to be kept constantly in mind. From the public health point of view, this is the crucial issue: to prevent dissemination of the infection, which is almost entirely a function of the primary, secondary and relapsing manifestation of the first few years. This fact indicates that the public health responsibility and the duty of practicing physicians come within the early months and years of the disease. It is to be hoped that the efforts of physicians throughout the country, combined with the splendid program of the U. S. Public Health Service will control and eventually stamp out syphilis to obtain the greatest security for the patient and the population.

Hysteria and Malingering

—The Differential Diagnosis from Traumatic Organic Disease of the Central Nervous System—

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Chicago

THE differential diagnosis between organic disease of the nervous system due to trauma, and hysteria and malingering is in the majority of instances entirely possible.* As you know, this problem is of extreme national import because of the legislative innovations, the economic aspect in regard to industry and, most important, the affected or involved person himself. How is it possible with our present scientific knowledge, means and methods of examination intelligently and honestly to separate these three conditions? It seems logical and practical first to classify an affection of the nervous system as being due to an organic (traumatic) lesion or a functional disorder. When this is done, then one should attempt to differentiate hysteria from malingering or simulation. This latter step may be extremely difficult, and may require considerable effort.

Organic disease of the nervous system due to trauma always produces some type of objective evidence of abnormality which can be found on a most careful, scrutinizing, and, if need be, an elaborate examination. Time does not permit of going into every detail regarding the various methods of determining the presence of organic disease of the nervous system. This can be found in any of the good neurological textbooks. From a practical standpoint it seems that paralysis, paresis, increased motion, sensory disturbances, cranial and peripheral nerve changes, nutritional states and electrical examinations afford the best possible means of determining the genuineness of an organic lesion as compared with a functional affection. To begin with, therefore, it is suggested that all traumatic cases be considered as possible organic injuries until such proof of the existence of such lesions is not found or substantiated. This latter is best done by a careful and scrutinizing examination of the central nervous

* Given before the Central States Society of Industrial Medicine and Surgery, Cook County Hospital, Chicago, December 4, 1936.

system for the purpose of finding objective signs and symptoms of organic disease.

Organic Paralysis

IN ORGANIC paralysis, which may be in the form of a hemiplegia, monoplegia, paraplegia, diplegia or quadraplegia, definite objective signs and symptoms are present which have been proved to indicate organic disease. If the paralysis be of an upper motor neuron type, one would then find an iron-pipe type of rigidity or, in some instances, a flaccidity together with increase in the deep reflexes of the involved limb or limbs. There will be pathological reflexes as well as true clonus. The cremasteric and abdominal reflexes on the side of the paralysis will be either diminished or absent. The gait will show a circumduction of the involved leg or a crossed progression if both legs are involved. The upper extremity will be adducted to the chest, the forearm flexed at the elbow, the hand flexed at the wrist, and the fingers flexed into the hand. There is a contracture in the upper extremity in most cases. The palpebral fissure on the side of the paralysis is usually wider than the opposite one and closing of the eye on the side of the paralysis is usually not as firm as on the opposite side. The naso-labial fold is lost or its prominence is less on the involved side. The angle of the mouth is usually not elevated or abducted, and is closer to the outer border of the tongue as compared to the opposite side. The platysma muscle on the involved side is always paralyzed. All the above signs are constantly present until recovery or improvement takes place.

Functional Paralysis

IN FUNCTIONAL paralysis it is very rare to find all four extremities involved at the same time. There is always a period of a normal state from the time of the accident until the development of the first subjective symptoms of paralysis. One is more likely to see in hysteria monoplegia, hemiplegia, one arm and the opposite leg and rarely both legs. Although a functionally involved person can move his lower extremities in bed he claims to be unable to walk. This is astasia abasia. When he does walk he either drags his leg, walks on the outer aspect of his foot, or walks on the toes of the involved foot or heel. There is no flexor contracture (can be passively overcome and if not one can feel the voluntary attempt to prevent its straightening). All the other objective signs of organic disease of the central nervous system are absent. The platysma muscle always contracts. These patients are emotionally and nervously unstable.

Organic Paresis

IN THIS group where there is no actual paralysis one always finds small signs of weakness of the involved extremities. These are Hoover, Grasset-Gaussel, swaying of the lower extremities when allowed slowly to fall to the bed from a flexed position at the knee, falling away, abduction or clumsiness of the upper extremity when both of the upper extremities are outstretched in front and the hands are rapidly opened and closed. There is a loss of associated movements in walking in the involved upper extremity. The palpebral fissure is widened and the naso-labial fold, angle of the mouth, and the platysma muscle may be weak.

In the functional group no such signs are present and its true nature is readily appreciated.

Increased Motion, or Hyperkinesia

HYPERKINESIAS are occasional features of organic disease of the central nervous system due to trauma. These may be a Jacksonian convulsion (unilateral), convulsive tic of the seventh nerve, facial spasm, generalized convulsions, tremor of paralysis agitans, athetotic movements, intention tremor and head nodding. It is not necessary here to describe these types of increased motion due to organic disease. Other signs of objective evidence of organic disease are found with the above symptoms.

Increased motion occurs in functional affections usually in the form of tics, tremors, hystero-convulsions and peculiar movements of the mouth associated with giving out of queer noises from the mouth. The tics are always stereotyped, imitable, course, repetitive, and never interfere with functions such as eating, chewing, shaving, etc. These tic movements are always bilateral and result from contractions of a group of muscles involved in normal conscious movements. If continued for any length of time the movements seem to change, due to fatigue. This never occurs in organic hyperkinesia. The functional tremor may be vibratory and rapid or marked by coarse oscillations of average rate. It is often indefinite and inconstant in character; sometimes slight, sometimes severe; at one time present, at another absent; but always considerably influenced by mental states. It may be generalized, unilateral, or limited to one extremity. Tremor may be stopped by diverting patient's attention elsewhere. If one fixes two fingers of a trembling hand the other fingers cease to tremble. The hystero-convulsions are readily appreciated in that their onset usually occurs in the presence of others and is brought on by some emotional stimulus. It is bizarre, the patient very rarely injures himself, falls to the floor carefully, and keeps his eyes closed. Any attempt to open them results in considerable voluntary effort on the part of the patient to keep them closed. The pupils are never dilated. Some form of yelling, screaming or talking is heard. There is no post-spell somnolence, cephalalgia or confusion. There is no pallor at onset of attack. There is no cyanosis or biting of the tongue.

The sensory disturbances of organic diseases of the central and peripheral nervous system are well known. These are hemianesthesia, unilateral trigeminal analgesia, radicular, segmental, spinal and peripheral nerve anesthesia as well as dissociation of sensation (central canal) and Brown-Sequard sensory loss.

The sensory disturbances of a functional affection are characterized by the following: The onset is sudden after some emotional excitement; the loss of sensation is usually partial and there is a hypesthesia rather than anesthesia; the hemianesthesia involves the face, trunk, limbs and special senses, that is, there is a diminution of loss of sight, smell, hearing and taste on the hemianesthetic side; tubular vision and loss of cutaneous and mucous membrane reflexes. The distribution is usually characteristic. This may be

in the form of the stocking or glove type, or it may involve both lower extremities up to the inguinal region or both upper extremities to the shoulder. Occasionally one arm and the opposite leg are anesthetic. When sensation is carefully examined and tested the involved areas will be seen to be related to no known organic pathway for sensation. There is an inability to relocate a tender point along the spine. The involved areas are not definite, and change from time to time.

Trauma to the cranial and peripheral nerves results in a definite type of paralysis which is characterized by clear cut objective signs.

Functional involvement of the cranial nerves usually confines itself to hearing and seeing. Functional involvement of the peripheral nerves, especially wrist drop, is common. Here the patient fails to appreciate the fact that wrist drop is due to a radial nerve injury which is characterized by inability to extend the hand at the wrist, extend the proximal phalanges, extend the thumb and move the hand laterally and medially. In a functionally involved patient one finds that he claims to be unable to carry out any kind of movement of the involved hand. This of course is inconsistent, because the muscles supplied by the median and ulnar nerves are still able to contract, that is, opponens pollicis, abductor pollicis, adductor pollicis, interoseii, lumbricales, flexors of all the fingers and thumb and pronators. No attempt will be made here to outline methods of examining for functional deafness and blindness.

Traumatic organic lesions of the nervous system always result in nutritional changes in the involved areas. These are color changes, atrophy, myedema, increased myotatic irritability and skin abnormalities. When a peripheral nerve is involved sufficiently to produce an anatomical or physiological interruption one will always find evidence of abnormal electrical reactions from a diminution in electrical excitability to a complete reaction of degeneration.

Having once determined the functional nature of the affection it becomes necessary to differentiate between hysteria and malingering. This at times may tax the ingenuity and patience of the most astute physician. Nothing, it may be said, resembles malingering more than hysteria; nothing resembles hysteria more than malingering. In both alike we are confronted with the same discrepancy between fact and statement, between objective sign and subjective symptom. The following features are some of the important differential points between hysteria and malingering in relation to trauma to the nervous system:

Hysteria

THIS is a fairly common condition; always occurs in an emotionally unstable person who deceives himself unconsciously usually without design or motive; the demeanor and behavior indicates a nervous instability and fear; he gives extravagant and bizarre comparisons to demonstrate the unusual nature of his case; does not choose his words and is not on guard; slips into inconsistencies; he loses sight of compensation in his effort to impress the doctor with the intensity and variety of his suffering; he likes examinations and more examinations, and the more minute they are and the more observers present, the better; he loves hospitalization, care, fuss and treatment; his symptoms are the same whether observed or not, are usually amenable to psychotherapeutic methods, and the onset of the subjective symptoms is usually hours or days after the original trauma.

Malingering, or Simulation

THIS is a rare condition; the patient consciously, voluntarily and deceivingly tries to reproduce symptoms which he either previously had himself or observed in others; he may, for the purpose of gain of some kind, simulate a disease which he does not have or unduly exaggerate or prolong minor symptoms which he has; the intention is to deceive; he purposely alleges loss of function or feigns all kinds of nervous symptoms; experienced malingerers may throw fits, simulate paralysis, complain of blindness and deafness; detection is usually difficult and in many cases continued observation and expert examination only can establish the diagnosis; he overdoes his stunts; his exaggerations are gross; resents examinations and will do anything to prevent them; resists all efforts at cure; when not observed he stops playing his role and at that time it is easy to determine the actual condition; occurs at once after an injury; repeated examinations and long periods of observation irk a malingerer and sooner or later he will slip in his act; may affect symptoms that are alien to the disease that he attempts to imitate; he sees less than the blind, hears less than the deaf and is more lame than the paralyzed.

In conclusion may it be said that whenever you are called upon to examine a patient who is supposed to have sustained an injury to the nervous system your first responsibility is to the patient. By this is meant that you must carefully examine the patient with the utmost kindness and indulgence for the purpose of determining the presence or absence of objective evidence of organic disease of the central nervous system. To examine him brusquely, rudely and superficially presupposes a mental bias and implies a tacit insult. If all medical examinations are carefully and honestly carried out, the time may not be far off when we will be able to stop the present shameful and undeserving attacks on industry and economics in general.

Resuscitation

—of the Apparently Dead from Electric Shock—

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HAVING met Professor Jellinek in Vienna in 1930, and hearing his talk on this subject, I became very much interested in it.* He had devoted over 30 years to this work. In fact, he had done so much that the University of Vienna created a chair for his department. His experience had covered over 4,000 cases in 1930, and he is considered an authority.

He uses the same method of treatment for drowning as for lightning shock; and he cites a case of veronal poisoning and cases which had stopped breathing on the operating table in which the treatment was successful.

* Presented before the Congress of Railway Surgeons, Chicago, November 7, 1936.

Statistics from the various states show that in 1935 in this country 630 lost their lives from electric shock, and 6,228 died from drowning. Many of the deaths from electric shock are through ignorance—as for example, some boys urinating on an electic rail were killed; some are from carelessness, and others from defective electrical appliances. Professor Jellinek cites cases in which doctors, after examining the victims and noting no respiration and hearing no heart sounds, pronounced them dead and made no effort at resuscitation; cases in which co-workers became frightened and ran away without even releasing the victim from the electrical contact.

But thanks to our public utilities and their valiant first aid crews, many of these unfortunates are now saved. The Western United Gas & Electric Company has very efficient first aid crews, headed and trained by Mr. Schmeiser, who respond to cases throughout our section and give most excellent service. The methods of resuscitation and first aid work are being taught to the various junior organizations, such as the Boy Scouts, Girl Scouts, etc. And I believe if the public generally could be informed on the essentials of the methods of this rescue work, with the dangers attending the releasing of the victim from electric contact pointed out, and how to avoid these dangers, many could be saved. If the witness of such an accident knows nothing of the methods, or from fright runs away, results cannot be expected. To get results, the victim must be released from the electric contact as quickly and safely as possible, and artificial respiration started immediately, as every minute may mean life or death.

Not all persons are equally susceptible to electric shock. Dry skin is more resistant than moist. A person well grounded will sustain much more damage than if standing on a dry board, or if sitting in a rubber-tired vehicle. An alternating current is usually much more painful than a galvanic, though this may simulate the alternating every time the victim jerks to free himself. A person not afraid, or anticipating a shock, is more resistant; whereas one afraid or excited, tired or hungry will receive more damage. The voltage does not determine the fatality, as 60 volts have caused death; and victims have been resuscitated who have been shocked by 120,000 volts. Likewise, the amperage does not determine the fatality. Jellinek has found the animal experiments very misleading in this respect.

THE pathology produced in the human being may be found in the central nervous system, as an edema with its resulting manifold symptoms; or in the heart as ventricular fibrillation, a hemorrhage, or a myocardial degeneration. In the lungs edema or vomited materials are found which caused death by suffocation. Any of the internal organs may show large or small hemorrhages. The blood vessels show their greatest damage at the point of entrance and exit of the current.

In the bones you may find pearls of calcium phosphate of pea size. Burns of the skin, soft tissues or bones seldom are infective or produce pus, but remain dry, slough, and heal perfectly smooth. The new skin shows good vascularity, is soft, and has no tendency to shrink. Horner's symptoms complex in which the eyes become sunken with loss of reaction to light, accommodation, and narrowing of the aperture, will usually show complete recovery in three weeks. Some of the late sequelae in injuries to the eyes are choroiditis, retinitis, optic atrophy, opacity of the lenses, and glaucoma.

The sensations experienced by the victim vary greatly. He may feel only a pleasant stimulating effect; he may have no pain even though conscious. One stated that he felt as though he were pleasantly floating in the air. Some feel tired, exhausted, have a tingling in the fingers, or muscle cramps. A man who had contact with 10,000 volts felt as though his fingers had gotten longer. Another man who had a permanent damage to his hand felt good enough to go back to work after a few minutes. Another man who had a 2,000-volt contact freed himself, went to the hospital and there became unconscious. A man whose head had a 22,000-volt contact felt sleepy for several hours. Another man who had contact with 220 volts galvanic current, slept for 18 hours. The victim may have many kinds of nervous symptoms as hysteria, hemiplegia, paraplegia, parasthesia, paralysis, bladder or bowel disturbances. One man had no bowel movement for eight days. Diabetes and jaundice have been produced; partial deafness, disturbances in taste or swallowing have been found. From most of the foregoing Jellinek has usually found a spontaneous recovery to occur in a short time. Burns may be superficial or deep but seldom become infective or produce pus.

Jellinek speaks of four methods of death. *First,* sudden death: The man may be found dead in a tetanic condition, standing, sitting or kneeling. *Second,* a slow death: A man had a 220-volt lamp in his hand, got a shock, cried out a few times, was not able to free himself, and died. (The first and second methods of death are the more common). *Third,* interrupted death: A 20-year old boy got a shock from a 220-volt hand lamp, was thrown to the ground, the current was cut off and he was apparently well, but after a few minutes he collapsed and died. *Fourth,* delayed death: A man 39 years old shocked by 220 volts was resuscitated, taken home, spoke to the doctor, stated that he felt well, but two hours later had a sudden death. A man 43 years old was shocked by 380 volts, was never unconscious, thought himself lucky, but 24 hours later died suddenly with pain in the cardiac area. Postmortem revealed a coronary thrombosis. Another case is cited in which a man had a 110-volt cord come in contact with his neck, and was never unconscious but died suddenly 12 days later from an edema of the brain.

Postmortem findings: 20% of the postmortems show no particular findings in the internal organs and have only the skin burns to suggest an electrical death. Postmortem examinations do not show findings in the internal organs which could be termed characteristic of electric death, whereas burns of the skin can be definitely proved as of electric origin, both macro- and microscopically. Some of the most common postmortem findings are: Edema of the brain, edema of the lungs, or vomitus found in the lungs due to too brutal a compression of the chest in using artificial respiration. Jellinek cites the case of a man who drowned in the bathtub. He was smoking while

taking a bath, dropped his cigarette, and in grabbing for it came in contact with a poorly constructed electric heater, was shocked and drowned in the tub.

Treatment

JELLINEK has used essentially the same treatment for the apparently dead whether from electric shock, lightning, drowning, veronal poisoning, or inhibited respiration on the operating table, with good results. He cites a great variety of electric contact cases. One, of great interest, is that of a woman 30 years old and her daughter three years old who were knocked down by lightning and thought dead. One hour later a doctor happened to stop at the undertaker's, saw the bodies, and, knowing the Jellinek method, he started artificial respiration on the mother while a farmer did the same for the child. After working about one hour both were resuscitated and remained alive.

The Jellinek method is about as follows: The victim must first be freed from the electric contact as quickly and safely as possible. In releasing the victim, good judgment and caution must be used, as illustrated by the following cases:

A man who was standing on a crane came in contact with a 380-volt lighting wire. Twenty men ran out, although a helper called to shut off the lights. Finally a locksmith turned off the current, but no one was there to catch the man as he was freed and he fell from the crane and died from a skull fracture.

A man who contacted a high-voltage wire called to his helper to turn the switch, which was done, but nobody thought about preventing his falling into a vat of hot sodium sulphate solution, and he drowned.

A man hanging on a 220-volt wire, and his coworkers ran to the telephone to call for help, instead of getting hold of his clothing and pulling him down; meanwhile, the man died.

If a man has his hand caught on a high-tension wire and you can reach him, Jellinek prefers to stand on a dry board for insulation and separate one finger at a time and shove a piece of cloth under the fingers as they are released in order to do less damage to the hand. At times he uses a heavy knife with an insulated handle to cut the wire or a heavy cutting forceps with insulated handles, or throwing a chain over the electric wire so that it reach the ground. In releasing a victim never get hold of his bare skin, shoes or wet clothing. You can take your dry coat, wrap it around the leg or arm and with a sudden pull break the contact. After the victim is freed from the electric contact, artificial respiration must be started at once, as every minute of delay lessens the chances of recovery.

The two methods of artificial respiration most used are the original Sylvester method as advocated by Jellinek, and the Shafer method which is used more in this country. Jellinek prefers the original Sylvester method without compression of the chest. If possible, he places the victim on a narrow table, as it is less tiresome for the worker, the head to be at the end of the table, face up. The head should be laid flat and not bent backward, as that would shut off the air passages. The tongue must be kept out by means of a forceps, or held out with a cloth by an assistant, or by running a pin or sharp-pointed stick through the tongue. All false teeth or foreign bodies must be removed from the mouth. Artificial respiration must be kept up rhythmically and continuously without interruption till breathing starts, or until positive evidence of death is noted, otherwise to continue for at least three hours.

Jellinek slips rubber sleeves over the arms to the level of the elbows. The sleeves have loops for the fingers and thumbs. The rubber sleeves permit a better hold, and by their elastic action promote circulation. The arms are brought upward above the head, a little outward and to below the level of the body, and then down alongside the body without an expiratory compression of the chest. These respiratory movements should be at about the rate of 24 per minute. While using the artificial respiration, watch for a movement of the Adam's apple, or the mouth, or an act of swallowing. As soon as one of these is noted, stop the artificial respiration long enough to see whether the victim continues to breath, as Jellinek has found that he can be killed if compression of the chest is made, as the heart is trying to expand. All other methods of stimulation may be administered by an assistant, such as inhalation of aromatic spirits of ammonia, slapping the face or body with hot and cold cloths alternately, rhythmic tapping over the heart, tickling the throat, shouting loudly to the victim, hypodermic stimulants, stretching the rectum, etc. Lumbar puncture is done if evidence of edema of the brain is present.

But Jellinek maintains that the chief reliance must be placed on artificial respiration. He cites many cases of resuscitation. A soldier who had been drowned in the Danube was taken our after 15 minutes and resuscitated after a half-hour of artificial respiration. Also, the wife of a doctor who had tried to commit suicide by taking veronal in 1904 was apparently dead, but after one hour of artificial respiration, recovered. He also mentions a case of Professor Billroth, who while operating on an 18-year old girl for a goiter, discovered that his patient suddenly stopped breathing. He started artificial respiration, kept it up for an hour, and quit. After walking out of the room his assistants, Doctors Salzer and von Eiselberg, started again and worked for another hour and half, and the patient recovered.

He cites many other equally spectacular cases, and I have wondered if many of us have not stopped our resuscitation efforts too soon. The method used most in this country is the Shafer, which I will describe but briefly, as Dr. Hart Fisher, who has worked for over 20 years in this field and has written a number of very fine articles, will discuss the paper and demonstrate both methods. In the Shafer method, *first*, place the victim on his belly or in the prone position, one arm straight up and the other bent at the elbow, with the head resting on the forearm or hand so that the nose and mouth are free for breathing. *Second*, kneel straddling the patient at his thighs, place the palms of your hands on the small of his back with the little finger over the lowest rib, your fingers and thumbs held parallel and tips of the fingers just out of sight. *Third*, with the arms kept straight swing your body slowly forward so that your weight is brought to bear on the patient. Your shoulder should then be over the heel of

your hand. This should require two seconds. *Fourth,* immediately swing backward to completely remove all pressure. *Fifth,* after two seconds repeat the movements. Twelve to 15 respirations per minute are recommended. Most authorities now, however, agree that 24 respiratory movements per minute are more efficient; and most authorities condemn the artificial respiration machines.

The later or subsequent treatment in electric accidents is likewise very important and at times fraught with considerable danger. In all cases of electric burns, whether superficial or deep, Jellinek advises conservatism. As stated before, they seldom become infective or make abscesses, and he advises never to be in a hurry with surgery. To illustrate: He cites a surgeon who dissected two fingers which were badly burned. The patient developed a hemorrhage a few hours later. The doctor ligated the radial artery to stop bleeding, and a few hours later this began bleeding. He then ligated the brachial artery, but again a hemorrhage developed and the patient died the next day from loss of blood. Hemorrhage usually occurs after the second week, and up to 10 weeks after the burn. The arteries become brittle and thrombi frequently develop on the intima. He cites a case of a man 31 years old who had a burn in the left axilla by a 5,000-volt current—four months later he became suddenly unconscious and developed a hemiplegia, probably cerebral embolism originating from the thrombus.

Electric burns do not tolerate long hot packs or baths, and a sloughing surface is best treated dry. Jellinek cites a case of a severe burn of the elbow in which the bones and tendons were much exposed, but by conservative treatment he obtained a complete recovery with full use of the elbow.

In all severe or deep burns the nurse should always have a broad elastic bandage at hand and in case of hemorrhage apply it as a compression bandage.

I might cite numerous other very interesting experiences, but space forbids. I hope however, that I have aroused sufficient interest in the work so that you may all return to your homes and teach the various methods of resuscitation to your organization, and the methods of release, but also the danger involved. Impress fully the necessity for starting artificial respiration at the earliest possible moment, that it must be continuous, rhythmic, and not be stopped until the patient breathes or is definitely dead, and that the method of resuscitation applies not only to electric shock, but also to the other forms of death above mentioned.

In conclusion let me emphasize a few points. The victim must be released as quickly and safely as possible. In releasing the victim use every precaution to avoid being shocked. As soon as the victim is released start artificial respiration and keep it up till the patient breathes, or for at least three hours before giving up. If using the Sylvester method, keep the tongue out and do not use compression of the chest. Remove false teeth or foreign bodies from the mouth. If you have been taught the Shafer method use it rather than experiment with a method you are not conversant with. Do not stop artificial respiration to call help, as it may mean life or death.

Discussion:

HART E. FISHER, M.D., F.A.C.S., Chicago: My interest in this subject started when I was an intern in New York City. I was called one day to a terminal which was being built and found there four men caught behind an electrical switchboard; and I was so impressed with the terrific degree of the burns of these men, and the conditions in which they were found, and the fact that nobody had done anything, that I then and there determined to study electrification hazards.

What Dr. Henry said about treating skull fractures and what Dr. Besley said in his discussion apply 100% to electrical shock cases, when you take into consideration that in many cases there are indirect results of electrical shock. We have people falling from high distances from shock, and sustaining a skull fracture as a result of the fall.

When a man contacts a current high up on a pole, the chances are that he has not received very much of the electrical current in his body, owing to imperfect contact. Hence he may be more easily resuscitated.

When men have been electrified they have had an electrical shock—a terrific molecular concussion of cells of the body with the result that they are shocked—and the psychological shock which comes on from the fear and emotion that are aroused following their return to consciousness is often marked. If one of these same persons has also sustained a skull fracture during the course of the injury and we then try to take him up to the x-ray room, or try to institute surgery, we then have overburdened the patient. The thing to do is to get such patients to bed, temporarily immobilize with splints; and do not take them to the x-ray room, whether they have skull fractures or other fractures, but keep them quiet. What Dr. Henry said about morphine applies here also. Where we have just electrical burns, which is not the same condition at all, we do morphinize because there was not much current passing into the patient.

There is one question which always comes up on this subject: What are the factors that determine the severity of the electrical shock? These are many, whether the current is high or low. In high-voltage currents there is no interval in between to mark which is high and which is low; when we say high-tension we mean about 2,500 volts. Such currents in themselves act more on the central nervous system and affect the centers of respiration. The low-voltage current, between 100 and 550 volts, has an affinity for the cardiac area, and that is where we lose the most cases, because the ventricular fibrillation makes it impossible to resuscitate the patient. We can also have a combination of respiratory and cardiac failure at the same time. A man in contact with a high-tension current will be affected in both the respiratory and cardiac systems.

Another factor that determines the severity is the nature of the current. We used to believe the alternating current more deadly than the direct, but this is not so. They are equally deadly. The intensity of the current cannot be determined because we do not know the skin resistance to these currents, and skin resistance determines the amperage entering the body.

Do the shape and size of the electrodes have anything to do with the severity? Sometimes they are large and sometimes they are small, and sometimes it depends upon the position in which they are placed. The larger the contact, the greater the current flow into the body. For instance, there is a contact of the left hand with the current, and of the right hand with the ground; naturally, this current will traverse the heart area.

Then we also have the conditions of wet and dry. When wet we have better contact, the resistance of the skin is lowered.

The main factor is the duration of current contact, regardless of Dr. Jellinek's statement that it does not have much to do with the severity of shock. The duration is the prime factor in producing death. I can state this definitely from my own experience. We had a case in which the current went in the right foot and out the left foot. The man was held in such a position that he could not get away, with the result that because he was held there five minutes he was killed.

The method of rescuing an electrified person is very important. In rescuing such a patient it is better to let him stay as he is a little longer until you are positive that you can rescue him without injury to yourself, and without dropping him back in contact with the current, as this reshocking is usually fatal. You can always break the current. Remove the victim's part that is the easiest

to get free from the current. Never touch a bare surface with your hands. We use one hand in rescue work. When you see our men working you will notice that they run to the scene of action with the left hand in a pocket, because they know that they must rescue with one hand and avoid the possibility of current traversing the cardiac area. Another thing is to be sure that you do not throw the patient back on the contact. Many people when contacted with an electrical current are pulled loose by rescuers and then, because of lack of instructions, are allowed to fall back and be reshocked and die. You must be sure you can remove the person safely.

One word further in regard to resuscitation. Use whatever method you can most efficiently. Shafer's method is easiest for us, and can be taught to the laymen. Doctors do not see these patients early enough to be of much value in the rescue, and laymen at the scene must give first aid. They must be rescued immediately, and given artificial respiration methods immediately and persistently. Many are apparently dead; but people have been resuscitated as long as eight hours after the shock, and we had one case in which we did the modified Shafer method for 48 hours. I speak of this because of the fact that performance of resuscitation does not do any damage to the patient's structure or organs. On the case just mentioned, autopsy revealed that after this prolonged period of artificial respiration there were no demonstrable lesions due to the resuscitation.

In closing I want to say that many of you have heard me speak on this subject year after year, and you may wonder why I still stick to it. I do it because there is no improvement today in the treatment of electrification cases. The increase in the use of electrical energy throughout the nation, and the increase in the population and in industrial work, make the possibility of elctrical contact more common. We are seeing such cases every day, and they are on the increase. It is for this reason that I keep emphasizing this method of resuscitation.

Cancer Development

from Chronic Irritation

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IN AN approach to the subject of cancer causation, the view, which is becoming more and more accepted, that the disease is composed of separate entities must be borne in mind. Up to 300 years ago the word "cancer" was an all-embracing term and included leprosy, tuberculosis, elephantiasis, venereal disease, granuloma, actinomycosis, blastomycosis, etc. During the seventeenth century the first three of these diseases were differentiated from that which was called cancer. The eighteenth century saw venereal disease, actinomycosis, and granuloma gradually being separated from the remainder; these were joined in the nineteenth century by blastomycosis and, later, by Hodgkin's disease.

With this knowledge of the past before us, and the realization that present day cancer has so many different clinical characteristics with varying degrees of virulence, even though the microscopic findings may be identical, and are of so many different types from the laboratory point of view, it is logical to assume that further division of the multiplex cancer mass may be the great discovery of tomorrow. In this differentiation lies our hope. The battle that is going on in this field may be likened to war itself, for one of the high points in military strategy, in an effort to defeat the enemy, is to divide his forces into sections and deal with each separately.

We cannot, then, be dogmatic with regard to cancer etiology. The fact that some diseases separated from the original cancer mass are of parasitic origin may lead one logically to suppose that micro-organisms have a role at least in a part of that which still remains. There are, today, two definite schools of thought: The outstanding leader of one group, Dr. Wm. B. Coley, whose monumental work with sarcomas is classic, believed that all cancer is of micro-organic origin; the other group discounts this theory and believes that the cause is due to metabolic change or fetal rests. Both these viewpoints may contain a certain percentage of fact. But the main point to be emphasized is that once the diseases that are incorporated in these two schools of thought are differentiated from one another and treated as entities, the whole question of etiology will not present such a seemingly hopeless problem as it does at the present time.

While there is much difference of opinion in all phases of this great subject today, one of the earliest conceptions of an origin of the disease—that of chronic irritation—is agreed upon by practically all present day workers in the field. Broussais (1772-1838) encompassed all disease in the class of chronic irritation and believed that tumors, benign and malignant, were forms of chronic inflammation resulting from chronic irritation. Virchow believed that every inflammatory or irritative process tends towards the formation of granulation tissue which serves as a soil for cancer development.

Chemical, mechanical, actinic, and bacterial irritation of the external regions of the body, where cancer may develop, and often does, is an industrial and insurance problem of real magnitude. Craver states: "The carcinogenic action of sunlight is startlingly evident in xeroderma pigmentosum. In certain cases of melanoma sunlight has apparently been the chief irritant of a mole." By animal experimentation it has been proved that repeated applications of coal tar (tar cancer) over a period of time on a given area, have a sequel in cancer production. When cancer is produced through long work in certain fields it is usual to consider it as having an occupational character, such as arsenic irritation, the by-products of coal, particularly tar, soot (chimney sweep's cancer of the scrotum), pitch paraffin, anthracene, etc.; the bituminous schists, mineral oil, petroleum, x-rays, radium, and cobalt. In the World War many Belgians who had become German prisoners and were put to work in paraffin factories, developed cancer of the hands.

External scar tissue may be constantly irritated by mechanical means to such an extent that malignant disease follows at the site. A scar that impinges against epithelial or glandular cells in any region may initiate a cancer. There are some who feel that a scar in the cervix is itself the parent irritant, while others contend that the cancer which follows is due to a chemical irritation as a result of the eversion of the mucous membrane of the cervix from its ordinary alkaline surroundings into the vagina with its acid secretion, thus causing a change in the environment of the cells.

Because of the wide possibilities of irritation, the mouth, tongue, and lip are common sites of malignant change. Constant and prolonged chewing of an irritating substance, such as the betel nut in India, the aggravation caused by a pipe or cigar when placed at a favorite site in smoking, jagged and decayed teeth, poorly fitting dental plates, imperfectly constructed crown and bridge work,

all may prepare the soil for the invasion of a cancerous process.

In an effort to prove that stasis of the secretions of the mammary gland has a direct relationship to the onset of breast cancer, Adair and Bagg, in their experiments, ligated the gland ducts of female mice at the nipples, or prevented the young from suckling, thus causing a retention of the secretions. Cancer of the breast followed, without involvement of the nipples. They concluded that "mammary cancer is due to breast stasis and the resulting irritation that follows the retention of stagnating secretions of the breast."

Ewing draws attention to the fact that in mammary cancer anatomical studies have long pointed to the importance of stagnation of secretions as the initial factor in atypical proliferation and fibrosis which appear in the earliest observed stages of the disease.

Some authorities have suggested the bacterial origin of glandular mastitis, while others believe that toxemia or trauma is the source. However, after careful investigation Keynes came to the conclusion that the cause is to be found in chemical irritation due to stagnation secretions and epithelial debris. It is interesting to note that 50 years previously, in 1878, Creighton arrived at a similar conclusion.

While these claims are undoubtedly based on fact, the writer believes that a toxic influence from prolonged intestinal toxemia may result in chemical reactions in the blood which cause a chronic irritation that affects the mammary tissue and tends to produce abnormal changes. The writer has had case after case of what he has denominated as "lumpy" breasts, because of the shot-like feel of the breast tissue, usually along the edge of the pectoral muscle where the mamma itself is suspended. These have returned to normal in many instances, when the gastro-intestinal tract has had remedial attention and the breasts, which are usually dependent in these cases, have been supported. Many patients who have consulted him for operation on the advice of their physicians, as cases with cancer, have had the so-called "cancer" disappear when the intestinal toxemia has been relieved. In large, dependent breasts, the engorgement of blood due to the elongation of the vessels, and decrease in their caliber, which results in resistance to return circulation and retention in the glandular structures of blood having a certain amount of toxicity, is often followed by cystic degeneration of the mammary gland. This process may be the forerunner of other pathological changes and cancer may supervene. The condition seems to differ from the blue dome cysts of Bloodgood, which do not, as a rule, develop into malignancy.

REPEATED trauma, or even one sufficiently severe trauma, especially to glandular tissue, has resulted in malignant changes. Referring to injury and breast cancer, the writer stated in one of his articles: "The processes induced by a single trauma may result in changes of the normal structure of the gland or of tissues in juxtaposition to it, and be followed by an irritating scar with a cancer as its sequel. This may be deep-seated as, for example, an organized blood clot in the breast; the initial swelling, tenderness, and any ecchymosis may disappear but deep in the structures, the fibrin forming part of the blood with the torn tissues, may amalgamate into an irritating scar. At this point cancer may and does, at times, occur." In other words, in the delicate gland structure, there is a continuing irritation from this scar.

Toxic conditions of the teeth, tonsils, sinuses, gall bladder, and other parts of the gastro-intestinal tract, have their effect on the glandular system. In citing McCarrison's theory of the cause of goiter, Sloan stated: "He believes that the seat of the infection is in the gastro-intestinal tract, and that he has cured endemic goiter by the administration of intestinal antiseptics and lactic acid ferments, and by vaccines prepared from the intestinal flora." In my own files are records of many cases of goiter which have been entirely cured by attention to toxic conditions, especially of the gastro-intestinal tract. In like manner, the mammary gland is also affected with tumefactions which disappear after the focus of infection has been discovered and treated.

Internal irritation, because of indefinite and mild symptoms before the onset of gross malignancy, is difficult to discover and often impossible to control. Ewing states that little has been proved with regard to the causes of gastric cancer, but there is strong circumstantial evidence pointing to buccal and gastric infection and intestinal stasis. In an article on carcinoma of the stomach, Hurst stated that 65% of such cases are secondary to chronic gastritis, as many cases of mammary and lingual carcinoma are secondary to chronic mastitis and chronic glossitis respectively. He believes that the majority of cases of chronic gastritis are due to the constant swallowing of irritating material from infected teeth and, though less frequently, from infected tonsils or nasal sinuses, to the excessive consumption of irritating foods and drink, and to the bolting of unmasticated food. He concludes that chronic gastritis—chronic irritation—is the most common precursor of gastric cancer.

In a personal letter to the author, Dr. James E. Davis, eminent Professor of Pathology of the Detroit School of Medicine and Surgery, writes: "You will note that a very noticeable percentage of these cases (hepatic cirrhosis) become carcinomatous. There seems to be no reasonable reason why, in the case of loss of parenchyma and replacement of loss of connective tissue following the effects of some outstanding toxic or inflammatory factor, such as may obtain in this disease, we do not have the same fundamental irritative factors present that so often precede malignancy in other tissues."

The field of physio-chemistry, which is more and more coming to the fore, may help solve many questions in the cancer problem. It is the belief of Alexander that "trauma sets free into the injured tissues exceedingly potent substances, which seem to be responsible for the *shock* due to injury. The question then arises: Can these substances set the stage for or produce mutation or modification in cells, leading to cancer?" He cites the experiment of Dr. Fenton B. Turck who, many years ago, under anesthesia, ligated a cat's paw, and then crushed it. No shock appeared. On loosening the ligation, however, so that the freed substances could be washed into the blood stream, shock promptly appeared. If the crushed paw was amputated before opening the ligation, there

was no shock. Alexander continues: "The molecules of the chemist and the genes of the geneticist are so near in size that we may hope to determine more closely, physically and chemically, what are the basic changes which underlie what the clinician recognizes as cancer."

In the question of cancer causation we are still on the highroad, with many by-paths to be traversed and investigated with experimental footsteps. By whatever other means malignancy may arrive to attack the human subject, there is one sure route agreed upon by all—that of chronic irritation.

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Skull Fractures

—Diagnosis and Treatment—

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"IF YOU can keep your head when all about you are losing theirs and blaming it on you" . . . .

Pope once said: "The only thing great on earth is man, but in man the only thing great is mind". If that is true, then the most interesting part of anatomy is the organ through which the mind manifests itself, or the brain.*

In no department of medicine have more far-reaching and fundamental discoveries been made than in that of the nervous system.

In the short time allotted me it would be unwise and impracticable to discuss anatomy and physiology; only the end results can be given as they apply to the practicing physician. The operating surgeon who keeps up with the experimental work being done is from three to five years ahead of the text books.

Caesar said: "All Gaul is divided into three parts". So this audience. Some have forgotten more about brain surgery than the essayist will ever know; others are his peers, and we meet on common ground; but there are still others who may take home useful information that will be life-saving.

Let us assume that you are in the latter class, and have come here to learn how to handle a little more skillfully the most delicate cases in all surgery.

First, reread our text: "Keep your head".

Like all Gaul, brain cases fall into three classes:

The first are the very mild cases which will cause no trouble or worry; they get well with little or no treatment.

The second are the very desperate cases that will die, no matter how treated.

And the third—and this class will tax your skill—are the ones whose recovery depends on you.

You can readily see that no hard and fast rules can be laid down to cover these three classes.

Now a word of warning about the first class—those cases that are just a "bump on the head". An injury to the brain which produces unconsciousness for half an hour may show up with marked mental changes or slow cerebral hemorrhages. Do not commit yourself that such an injury is of no importance—later it may embarrass you.

The second class of cases are usually so outstanding as to require no special skill in order to recognize them at once. But treat them in the most humane manner.

And now for the third class—those who have a fighting chance. Probably the most discussed question is which cases should be operated, and when. The patient who has a compound comminuted fracture should be immediately operated. These cases still follow the dictim of Fenger: "All cases of septic meningitis die". Fenger was a Master—his dictum will hold as long as infection invades the cranial cavity.

In these compound fractures, the first dressing put on within half an hour after the accident is more important than all the skilled work of the brain specialist who comes on the scene the next day. Little or nothing can be done after meningitis has set in. Proper dressing is so important that, even in shock, it should be attended to at once.

Without belittling the x-ray in head injuries in any way, it seems to the writer that it is a mistake to make a frantic rush to the x-ray department and demand an x-ray, as if nothing could be done until a fracture is shown. Usually the fracture is the least important part of the whole picture. Even a depressed fracture may not be, and usually is not, of prime importance. Some things should be done routinely in this third class.

A careful physical examination of the entire body should be made. The unconscious patient cannot call your attention to the broken or dislocated bone. The shock may be due to a ruptured spleen. The head injury may be a minor thing compared to a ruptured bladder, bowel, or liver.

So we repeat that a careful, complete physical examination should be made. The blood pressure, pulse, respiration, and degree of coma should be taken every four hours.

The value of spinal puncture has been greatly over estimated. It is probably better to have one done, as it does give a certain amount of information. The idea of reducing brain pressure by

* Presented before the Congress of Railway Surgeons: Meeting of the Association of Surgeons of the Chicago & North Western Railway, and the Burlington Surgeons, Chicago, November 6, 1936.

spinal puncture is almost if not quite a delusion. If a spinal puncture is useless as a therapeutic measure, a decompression is infinitely worse. This is the reason: To do a temporal decompression that amounts to anything, a large opening must be made and the dura split. If the brain is under tension it bulges out like a hernia, and that part of the brain so herniated is permanently traumatized and in time to come may produce more serious mental trouble than the original injury. Put it down along with the point about septic meningitis—a decompression always does harm and never good.

But there are two other types of cases which should be operated—hemorrhage of the meningeal artery, and decompressed fractures. In and of itself, hemorrhage of the meningeal artery will not produce severe shock and can be attended to in the first two or three days. Depressed fracture in and of itself does not produce shock. The surgeon can choose his own time to elevate the depression, but it would be a mistake to leave this indefinitely. Some men say: "If it is in a silent area, let it alone". There are no silent areas of the brain. There are some areas of whose function we are ignorant—but silence and ignorance are not synonymous. It is quite possible that these so-called silent areas when injured may rebel against insult quite as vigorously as better known areas.

It is startling to note the number of cases that have been given morphine before being brought to the hospital. The average practitioner apparently does not realize the depression morphine causes on the respiratory center, or the fight that the brain surgeon puts up to keep pressure off of the basal ganglion. The point then to remember is no opium in brain trauma.

Cerebral edema is the cause of death in many cases of brain injuries. Glucose, sulphate of magnesia or hypertonic salt solution are far superior to surgery in controlling edema. Any of the barbital family are much safer than opium for delirium or restlessness.

SUMMARY: Operate at the proper time:
Compound comminuted fractures,
Meningeal hemorrhages, and
Depressed fractures.
Do not give opium.
Do not do decompression operation for edema.
Do a complete physical examination.

Discussion:

FREDERIC A. BESLEY, M.D., F.A.C.S., Waukegan, Illinois: Dr. Henry has presented his subject admirably, and has based his conclusions upon accurate observation of the patient as a clinical entity and not upon academic reasoning.

The problems involved in the care of a skull fracture are not yet solved to the entire satisfaction of the whole surgical profession. There are two schools of thought relative to the question of dehydration and the maintaining of the water balance. It is axiomatic that every case is a law unto itself. Many years ago I had the opportunity of doing autopsies on 100 cases, and it was very apparent that the anatomic conditions were never the same. Therefore there can be no accurate standardized method of treatment evolved as applicable to every case.

Some of us feel that the dehydration method is often carried too far in the individual case, and should be used cautiously and with discrimination. It may do harm, particularly in early cases where the intra-cranial pressure is controlling continuation of hemorrhage. Spinal punctures are not without danger, and should be used with great caution.

Certain procedures should be followed in all cases, granting that a reasonable diagnosis has been established. Don't take an x-ray immediately. It is of little value. Place the patient in bed and treat any shock that may exist. Carefully check blood pressure, temperature, pulse rate, and particularly increasing restlessness or deepening coma. These observations should be made frequently.

Dehydration may be used in some selected cases. If the pulse rate rises rapidly, if the temperature reaches 103°, if the blood pressure rises and coma deepens, then do a subtemporal decompression—one or both sides—as the best known method of directly reducing intra-cranial pressure.

In the present state of our knowledge we should keep an open mind and be loath to use too radical procedures. Sometimes our conclusions relative to results from some specific form of treatment are based on impressions and not upon fact.

Abdominal Trauma

—and Its Diagnosis—

McMICKEN HANCHETT, M.D., F.A.C.S.,
Council Bluffs, Iowa

NON-PERFORATING abdominal trauma may be in the nature of (1) simple contusion or concussion, or (2) contusion with visceral damage.*

SIMPLE CONTUSION OR CONCUSSION of the abdomen usually presents the classical picture brought to mind by the expression "having the wind knocked out." Following an abdominal trauma, the patient will be in a state designated occasionally as "sympathetic or abdominal shock." The face is pallid, expression anxious, the skin is clammy, respiration shallow and rapid, temperature subnormal, pulse rapid and of low pressure. The pupils are dilated and eyes expressionless. Nausea and vomiting usually occur. The abdomen usually shows a protective rigidity, general or local. Peristalsis may be momentarily decreased or absent, depending upon the severity and extent of the trauma. Pain is an early important symptom. In extremely severe cases associated with unconsciousness or impending death, defensive rigidity may be lacking and normal muscle tone may be entirely gone.

If there is no visceral injury, the patient is most ill immediately following the accident, and as recovery from shock occurs symptoms decrease. The pulse becomes stronger and the rate drops, color returns, the abdominal rigidity relaxes, the pain disappears and is replaced by soreness. This condition requires rest, quiet, heat, and withholding everything by mouth. The administration of stimulants and hot drinks is dangerous because of the danger of hollow viscera injury. These cases must be watched intelligently and closely, as serious intra-abdominal damage may be present even though the trauma may have seemed trivial.

A case in point is that of an 11-year old boy who was brought to the hospital late one afternoon. When he reached the hospital he was dead. He had been kicked in the abdomen by a colt 27 hours before and the "wind knocked out of him." He was seen by the family physician soon after the accident and was thought to be only slightly injured. The subsequent vomiting and increasing pain were thought by the family to be natural sequelae. He remained in bed without particular attention until about 26 hours after the injury, when his grave condition was so apparent the

* Presented before the Congress of Railway Surgeons: Meeting of the Association of Surgeons of the Chicago & North Western Railway, and the Burlington Surgeons, Chicago, November 6, 1936.

family became alarmed. He probably died before he was taken from his home. Autopsy revealed a complete transverse division of the jejunum about 20 cm. from the duodenojejunal juncture.

The signs of this visceral injury for which we must be on the alert should not be those of a fully developed peritonitis or of a terminal anemia. They should be the early, warning signs, following an initial improvement from the original shock. Probably the earliest and most reliable index is the pulse—increasing in rate and decreasing in force and volume. Persistent rigidity, if accompanied by nausea and vomiting; an increasing anemia, especially associated with an increasing leucocytosis and absent or decreased peristalsis, are the signs indicating a serious condition.

CONTUSION WITH VISCERAL DAMAGE may result from trivial or severe trauma. The character of the force is extremely variable and it may be applied in many ways. These are important in determining the viscera affected. In general, it may be said that trauma applied to a circumscribed area is more apt to injure the gastro-intestinal tract and kidney, while force which is applied more diffusely over a wide area is more liable to injure the liver or spleen. Engorgement during physiological activity or pathological changes in the organs to which the force is applied indirectly may predispose to injury. The viscera of the young and those which have greater mobility are not as frequently injured as are those of the old or those which because of their fixity cannot change form or location readily when force is applied.

The order of frequency in which the various intra-abdominal viscera are involved by non-penetrating trauma varies. It is very evident, however, that the solid organs are affected, either singly or combination, very much more frequently than the hollow viscera. In 140 cases reviewed by Lewis, of which 95 were analyzed, 75% involved solid viscera. Beekman, reviewing 59 cases, found 60% involving solid viscera and 10% hollow organs. In 84 cases reported by Hinton, 78% were injuries of solid organs and in 22%, the hollow viscera were involved. Other reports agree with these, indicating the greater frequency with which the solid organs are involved.

In massive intra-abdominal damage, the patient is frequently moribund, and the general diagnosis is apparent. In the presence of such profound shock, the indications are for general supportive treatment. Where there is some degree of recovery following the initial shock, we come to the critical period in the management of these cases. It is necessary to determine whether or not there is intra-abdominal visceral damage. This has been called the "latent period" and it is at this time that the type and extent of the intra-abdominal injury must be studied. A continuation or return of nausea, vomiting, increased pulse rate, rigidity and pain and leucocytosis are the general signs indicating serious condition.

This study is not only important as a guide in selecting those cases requiring operation, but also in choosing the manner of approach. While making this study, it should be kept in mind that retroperitoneal hemorrhage, injuries to the chest and diaphragm and some compression fractures of the upper lumbar vertebrae frequently simulate intra-abdominal injury. Extra-peritoneal bleeding, while rarely massive and usually self-limited, produces in addition to symptoms of concealed hemorrhage, those of peritoneal irritation, namely, nausea, vomiting, pain, rigidity and diminished peristalsis. Examination of the chest may reveal fractured ribs and hemothorax or ruptured lung with pneumothorax. Localized tenderness along the spinous processes suggests the possibility of fracture of the body of one or more vertebra. X-ray is of use later as a confirmatory method. In damage to solid intra-abdominal viscera, generally hemorrhage within the abdomen is the outstanding cause of symptoms. Special factors tending to indicate which of the solid organs may be involved may be taken up separately under those organs.

LIVER damage is usually the result of:

1. Blows by a broad surface on upper abdomen, especially right side;
2. Falls from a height landing on buttock, feet or abdomen; or
3. Being run over. (Eliason says—from right to left). If after such a contusion, signs of hemorrhage present themselves, associated with tenderness and rigidity in the right upper quadrant, suspicion should be directed to fracture of liver. In addition to these symptoms, Finsterer has observed a bradycardia and pain referred to the right shoulder has been described. The common lesion found is a stellate or Y-shaped laceration of the right lobe, though a subcapsular rupture is described by Bradenbergh.

SPLEEN: Injuries to the spleen result from trauma to the left upper abdomen of a crushing nature, falls from a height, and occasionally from a rather trivial injury. Owing to its position under the ribs, it is more often injured in association with injuries of the liver or kidneys than alone. Shock and evidence of profuse intra-abdominal hemorrhage are the two conspicuous features. In addition, one finds localized pain, tenderness and rigidity, in the left upper quadrant. Pain referred to the left shoulder, painful, jerky respirations and restricted diaphragmatic movement on the left, confirmed by x-ray, are often aids in arriving at a diagnosis of ruptured spleen.

PANCREAS: Isolated injury of the pancreas is quite unusual, and in my experience has been limited to one case. A young man of 20 years was kicked over the epigastrium by a horse. He was seen by his own physician and the injury was regarded as of a trivial nature. After a day or two of rest, he again pursued usual work on a farm. About two weeks later he noticed that his belt was too tight and soon observed that his abdomen was larger than it had been. He again consulted his physician who found a large cystic tumor in the epigastrium. At operation it was found to be a cyst, having its origin in the head of the pancreas. Apparently this was a crushing injury to a part of the head of the pancreas without rupture of the capsule. Marsupialization of the sac was followed by complete recovery.

KIDNEY: Rupture of the kidney is a common injury and occurs most often from trauma in the loin. The symptoms of hemorrhage appear slowly and usually are not of a serious nature. If the rupture is extraperitoneal, which is more often the case, the signs of peritoneal irritation are insignificant at first. As previously stated, these are

suggestive of intraperitoneal lesions. These findings are usually associated with hematuria, and at times urinary frequency and pain. An x-ray will often help in making the diagnosis. The iliopsoas muscle line will be obscured on the side of the injury and frequently the gas containing viscera will be shown to have been displaced mesially by the hemorrhage posterolaterally. If the laceration extends through the kidney and involves the pedicle, instead of a slowly progressive condition, one is confronted with the signs and symptoms of profuse intraperitoneal hemorrhage.

Rupture of the Hollow Viscera may include any of those within the abdominal cavity. However, except when associated with local pathology, or injury of other abdominal organs in massive crushing injuries of the abdomen and chest wall, rupture of the stomach and gall bladder are uncommon, due to their relatively protected location. It must always be kept in mind that gastric ulcer may be present without previous history. In a like manner, late rupture of a diseased gall bladder may occur following comparatively slight localized trauma. The intestine occupies the greater and more exposed portion of the abdominal cavity, and is therefore more frequently injured. Also, as would be expected, the jejunum and ilium, being more centrally located, are much more frequently involved than the duodenum and colon. Cooke collected 700 cases of rupture of the intestine, in which 82% involved the jejunum and ilium about equally and 18% were equally divided between the duodenum and colon. Lockwood in 652 cases reports about the same experience.

The condition may be due to crushing injuries, sudden compression as in automobile accidents and train accidents, a sharp blow on the abdomen by a relatively small object as a kick by a horse or man, and compressed air being suddenly forced into rectum. Except for the last type, the factors affecting the injury are either crushing the intestine against the vertebral column, or the bursting effect of gaseous and liquid contents incarcerated within a loop of bowel. This latter mechanism, first suggested by Schonleber, seems to explain the lesion found in those cases resulting from rather slight injury.

The symptoms of intestinal rupture due to external violence vary with the severity of the injury, the association with other injuries, the location and extent of the lesion of the bowel, the condition of the bowel when injured—whether full or empty—the interval and activity of the patient following the injury, the treatment from the time of the accident until examined—whether or not food or fluids have been given by mouth and whether or not morphine has been administered. Symptoms may appear immediately following the injury or be delayed for hours or even days, even though complete rupture has occurred.

Moynihan, LeConte and others have pointed out the difficulty in recognizing early, a rupture of the first part of the jejunum in a patient who has not eaten for many hours. Fifteen hours may elapse before marked symptoms appear in such patients. Holland, Poland, Battle and Berry have reported cases with incomplete rupture who went for days, weeks and even months before complete rupture occurred followed by grave sequellae. In the majority of cases, however, while the clinical picture as a whole may not be present, certain symptoms point to the condition and demand the closest attention.

Primary Shock: Primary shock develops in most patients immediately after rupture, but may not be marked if only rupture occurs. However, it may not occur at all, develop late or rapidly clear up and later the patient pass into secondary shock. Complete rupture or extensive laceration may not produce marked primary shock, and a single small perforation may result in grave shock. Apart from the effect of hemorrhage, the speed with which reaction from shock follows is no indication of the extent of the lesion. Berry and Guisseppi reported that in at least six out of 59 cases, primary shock did not occur, and the patients walked home or to the hospital. Christopher and Nadler each have reported one case in which the patient walked home. Curtis reported two patients without any marked symptoms for 24 and 26 hours, respectively; 80% of his patients, however, developed primary shock.

Additional symptoms are:

1. *Facies abdominalis*: The pale, anxious, drawn face, and the alert and apprehensive expression of the patient with ruptured intestine is almost pathognomonic. It usually appears quite early. Later it gives way to a peculiar listlessness, which is indicative of the profound toxemia of a peritonitis.

2. *Temperature and pulse:* Following recovery from the subnormal temperature and rapid thready pulse of the initial shock, the temperature usually returns to normal and the pulse decreases in rate and increases in volume. Elevation in temperature is not an early sign and rarely rises much above normal until a well developed peritonitis is present. An increasing pulse rate, however, following recovery from the initial shock, must always be regarded as an important warning. It is one of the most dependable early signs and demands painstaking thorough investigation.

Vomiting invariably occurs. It rarely appears at first except in a lesion of the duodenum or first part of the jejunum. It usually develops within an hour or two of the injury, depending upon the degree of primary shock and the reaction to it. Nausea is frequently present from the outset. The vomitus is rarely blood stained except when the duodenum is involved. Later it is stercoraceous.

Pain and tenderness may result from superficial bruising only, but there is a deep, localized type of pain, and particularly deep tenderness, present in practically all cases. This varies with the site and extent of the lesion, the state of the bowel, and the reaction of the surrounding tissues to infection.

Abdominal rigidity localized to the area of injury or general, is present in all cases, except a small percentage in whom the "flaccid abdomen" is found. The latter is a grave sign, usually indicating extensive multiple lesions of the small bowel, ileum, in a patient with little general resistance, and warrants a grave prognosis regardless of treatment. Progressive board-like rigidity is usual and indicates serious trouble. Absence of abdominal movement on breathing suggests the type of injury. Dullness over the area involved is as a rule late evidence of nature's effort to wall off the area about the rupture.

Abdominal distension and tympany may be due

to paresis from trauma apart from perforation. Increasing distension may be due to bleeding, escape of fluid content of the bowel or to serous fluid poured out by the inflamed peritoneum. Its appearance eight to 10 hours after the appearance of the first symptoms is a grave sign. Like *absent liver dullness,* it is late evidence of serious trouble. LeConte, Moynihan and others have pointed out the worthlessness of both of these signs in the early diagnosis of ruptured intestine. They are observed in late cases only or those that are inoperable for other reasons. In my experience, these patients do not survive operation.

Dullness in the flanks is another undependable finding. If present early, it invariably is due to hemorrhage. Intestinal content rarely escapes even in complete rupture during the first six hours (Lockwood). Dullness appearing late in the absence of hemorrhage, is usually due to serous exudate and is a grave development.

Surgical emphysema is not an uncommon occurrence as a result of rupture of the bowel and invariably is due to a rupture of the duodenum or colon retroperitoneally. It suggests a grave type of lesion because of retroperitoneal leakage and infection.

X-ray. The presence of free air or gas in the peritoneal cavity is the pathognomonic sign of rupture of a hollow viscus. The only positive method of determining this is by x-ray. If possible, this should be done in the standing position. By this method there can usually be demonstrated a layer of air between the diaphragm and the liver. If the condition of the patient does not permit the standing position, films taken in both the lateral and prone positions may show a shifting gaseous bubble.

To SUMMARIZE: Diagnosis of ruptured intestine is suspected in those patients giving history of trauma, in whom, following recovery from the initial shock, there is present: (1) facies abdominalis, (2) increasing pulse rate without other signs of profuse abdominal hemorrhage, (3) vomiting, (4) increasing deep pain and tenderness, (5) persistent abdominal rigidity, (6) leucocytosis. X-ray confirms the diagnosis.

Rupture of the urinary bladder may be either intraperitoneal or extraperitoneal. The great majority of extraperitoneal ruptures are associated with fracture of the pelvis. Compression force or a sudden blow over the suprapubic region, when the bladder is distended, will frequently result in a rupture. The patient at first may be but slightly disturbed by this injury. Only after a few minutes will pain be manifested. Strangury, bloody urine, and pain and tenderness appear. Catheterization will yield either a small or abnormally large amount of bloody fluid. The introduction of a measured amount of fluid through the catheter to make certain of the diagnosis is rarely necessary and is not a safe procedure especially if intraperitoneal rupture is suspected. Intraperitoneal rupture will give the symptoms of a rapidly spreading peritonitis, though Lewis cites instances of remarkable delay in their onset. Extraperitoneal rupture is evidenced by swelling, tenderness and pain in the suprapubic region and as extravasation of urine takes place, the swelling extends to the inguinal region. At times, symptoms of fracture of the pelvis may overshadow or conceal those of a bladder injury.

The diagnosis of the conditions associated with subcutaneous intra-abdominal injury has more than the mere academic interest. It is necessary in order that we may definitely determine the procedure.

These cases divide themselves roughly into two groups: (1) those in which conservative non-operative measures may be followed, and (2) those in which early operation is imperative.

Non-operative measures, I believe, are the methods of choice in laceration of the kidney and liver, except in the presence of excessive hemorrhage. Of 18 cases of ruptured kidney, reported by Hinton, 17 cases were treated without operation and 16 recovered. The one case that died was suffering from multiple serious injuries and died in 24 hours. Lewis reports 9% mortality in ruptured kidney managed conservatively, as against 40% subjected to operation. O'Connor at this meeting last year reported much the same experience. Wagensteen, Beekman and others agree.

There is not quite the same concurrence in the management of rupture of the liver. Hinton, Beekman and others point out the lack of active bleeding in those cases operated, except in instances of such extensive liver and vessel damage that neither packing nor suture was able to control the hemorrhage. This has been my own experience.

Conservative treatment consists in (1) *rest and supportive treatment,* and (2) *transfusion.*

There is an element of danger in the use of blood transfusion in cases of this type. Blood pressure has fallen, due to the intra-abdominal hemorrhage and this lowered pressure is an important factor in controlling the bleeding. It is therefore, quite important that large transfusions be avoided and small transfusions given slowly. If the blood pressure be raised suddenly, fresh hemorrhage may ensue. Not more than 250 to 300 cc. of citrated blood should be given at a time. This may be repeated if necessary. Hinton suggests gum acacia solution—6% gum acacia and 20% glucose in 500 cc. saline. This solution should be given (102°-105°) at the rate of 4 cc. per minute, requiring two hours for 500 cc. This has proved satisfactory in my hands.

Operation is imperative in all ruptures of hollow viscera, ruptures of spleen, and those of liver and kidney with signs of profuse hemorrhage. The choice of time of operation is most important. It is obvious that during profound initial shock, the added shock of any operative interference is contra-indicated. Following recovery from the primary shock is the period of "optimum opportunity." Prompt action at this time will increase our satisfactory results.

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Discussion:

E. P. HELLER, M.D., Kansas City, Missouri: Dr. Hanchett has left relatively little to be said in the matter of diagnosis of intra-abdominal trauma which is non-penetrating. The diagnosis after all is the problem that confronts us without really the need for great haste, except in those cases where perforation or hemorrhage is obvious. In those cases, of course, even in the presence of shock, operation is justified. In the absence of definite evidence of those two conditions we should really use discretion and take time for a definite diagnosis.

Several patients of this type have been seen at the Kansas City General Hospital; some of them came in with penetrating wounds and injuries and the diagnosis was rather obvious. Those patients who are desperately injured with penetration of the abdominal wall and vessels are sent directly from the receiving ward up to surgery, whereas those in whom there has been some injury of a non-penetrating type are sent to observation ward.

I recently had statistics gathered in order to judge what proportion of these injuries were non-penetrating. In a period of three months 608 patients were received in that ward; 63 of them head injuries and seven of them non-penetrating abdominal wounds, and none of them had abdominal wounds of a nature that required operation. At the Research Hospital, in 4672 cases in 1935, there were one ruptured bladder due to fracture of the pelvis, and two ruptures of the large bowel, and two of the liver and spleen. That gives an idea of the proportion of abdominal injuries in a private and public hospital. The 608 cases mentioned were taken care of in the observation ward, with few exceptions.

As to the use of morphine in obscuring the diagnosis, the pendulum has been swinging to the use of morphine early because it is now felt that it is more likely to localize the diagnosis; it is indicated to combat shock. It seems to me that one of our chief duties is to have a routine to follow in the care of these patients. It has been our habit in patients in whom there are multiple injuries to order the patient to bed; a catheterized specimen of urine is secured; stool is obtained, with saline enema given to obtain one if necessary; blood is checked for hemoglobin and red count, and all data possible are gotten, including blood typing for transfusion. All this is done at the time the patient is admitted, and in that way we have something to go on in case of intra-abdominal hemorrhage, or in other conditions.

Preventive Medicine

Book Review by

C. O. SAPPINGTON, M.D., DR.P.H.

THE Fifth Edition of the above-named work by Mark F. Boyd, M.D., M.S., C.P.H., member of the regular Field Staff, International Health Division of the Rockefeller Foundation, contains new material on various subjects, among others: colds, ringworm, psittacosis, diphtheria, encephalitis, poliomyelitis, pneumonia, tuberculosis, malaria, typhus and relapsing fever; recent developments in sewage treatment are described; there are sections dealing with mottled enamel; and more extensive consideration is given to the vitamins, silicosis, and recent data relating to general morbidity incidence.

In the section on Occupational Diseases, there is a tabulation of the general hazards encountered in principal occupations, and an outline showing the principal industries in which lead poisoning may occur. Besides lead poisoning, consideration is given to arsenic, mercury, benzol, brass founders' ague, carbon monoxide poisoning, pneumoconiosis, silicosis, and phosphorus poisoning.

It might be well to emphasize the importance of general public health work and its phases to the general practitioner and especially to the industrial physician and surgeon. More and more will there be overlapping between the detailed consideration of public health questions and problems and specific difficulties encountered in the field of industrial medicine and surgery. As Dr. Boyd states in the foreword: "The medical profession can and must play an important role in the field of preventive medicine and public health. At present physicians are neglecting their opportunities. If this neglect continues, the opportunities will lessen and the field will be taken away from the physicians by a changing public sentiment. If these pages will bring home to medical students and practitioners a realization of their public health responsibilities and stimulate cooperation between public health authorities and the medical profession, the labor of their preparation will have been abundantly rewarded."

The fifth edition of this work contains 561 pages, was published in 1936 by W. B. Saunders Company, Philadelphia and London, and is priced at $4.50 in cloth.

Diseases of the Respiratory Tract

Book Review by

C. O. SAPPINGTON, M.D., DR. P.H.

THE above title is the caption applied to a volume containing the lectures given by 21 distinguished contributors before the Eighth Annual Graduate Fortnight of the New York Academy of Medicine.

Of particular interest to the industrial physician and surgeon is the lecture by Dr. Leroy U. Gardner on "Pneumoconiosis with Particular Reference to Silicosis and Tuberculosis." Dr. Gardner's opening statement is worthy of quotation: "The term 'pneumoconiosis' was coined to described the pathological *condition* of the lungs induced by the inhalation of dust. Recent usage has extended the original implication, and one finds many writers referring to pneumoconiosis as a *disease*. In certain instances it may be a disease but this is only true where the dust causing it is specifically irritating. Quartz or free silica, and the silicate of magnesium, asbestos, possess these properties. As far as is known today pneumoconiosis produced by other types of dust is no more a disease than the calluses on the palms of the hand. In fact many dusts seem to cause only pigmentation of the lungs without appreciable cellular reaction. Where slight changes do develop they resemble and are no more marked than those incident to advancing age. It is of course possible that other injurious types of dust may be discovered as more industrial surveys are made, but up to the present time evidence of significant reaction to non-silicious dusts is entirely lacking. A series of experiments is now in progress to determine how far various representative silicates may be capable of producing reaction in animal tissues."

The volume contains 418 pages and 56 illustrations, was published in 1936 by W. B. Saunders Company, Philadelphia and London, and is priced at $5.50 net in cloth.

Pre-Employment Examinations

—A Digest of the First Thousand of a Series—

MICHAEL H. BARSKY, M.D.,
and
JULIAN WOLFF, M.D.,
New York City

THESE examinations were made for a general contractor on an open cut job, requiring excavation, drilling, concrete work and road building.

All of those examined had met the requirements of the personnel department and presumably were fit and able to do their respective work; all to be employed were examined, and all subjected to the complete examination.

The immediate purpose of these examinations was to obtain a record of the physical condition and exact medical data as to the presence of such infirmities as would make employment and continued dust exposure inadvisable. The examination was conducted away from the job, at the Doctors' offices. Detailed occupational and medical histories were elicited by the examining physicians who took full cognizance of the natural tendency of the applicant to minimize, if not entirely omit, any complaints, previous injuries, or illnesses.

At least one six-foot x-ray of the chest was taken and studied in conjunction with the examination. Where indicated, check up re-examinations were made by other members of the medical staff.

The average time per examination was 45 to 60 minutes.

At the completion of the examination, based upon the findings, the applicant was rated into one of four classes, A, B, C and D, and thus there was established an approximate grading of his general health, which is used as a working index.

Class A—Those who were of average health and physique.

Class B—Those subnormal, owing to the presence of one or more conditions which, either of themselves or because of their potentiality, make their employment an undue hazard.

Class C—Those having one or more major conditions which made exposure incidental to this work inadvisable.

Class D—Those having silicosis, thereby contraindicating further dust exposure.

For convenience, the subject matter is studied under the following captions:

HISTORY:

Nativity. Race. Age.
Occupation.
Industrial history, solely as to dust exposure.
Previous pre-employment examination.
Family history and present complaints.
History of preceding defects and diseases.

EXAMINATION:

Weight. Vision.
Blood pressure. Teeth.
X-ray findings.
Abnormalities and diseases found.
Basis for classification—as to the specific causes.
Comparison with other groups.

The findings are recorded by tables for the entire group, with subdivision as to "Class" and are supplemented by explanatory text, where required.

ONE THOUSAND males were examined. The tables which follow contain the actual figures —the percentages are obvious. Tables 1 to 8 cover the history, the remainder the examinations.

NATIVITY is shown in Table 1.

	Class A	Class B	Class C	Class D	Total
United States	376	41	21	6	444
Italy and Austria*	215	29	10	13	267
Ireland	116	16	5	2	139
Scandinavia**	57	8	6		71
Great Britain	21	4	1	1	27
Poland	10	1		1	12
Germany	5				5
Greece	3		1	1	5
Russia	2	1	1	1	5
All Others	23	1		1	25
	828	101	45	26	1000

*Grouped with Italians, as they were of Italian extraction.
**Includes Norway, Sweden, Denmark and Finland.

Table 1. Nativity

The native born, Italians, and Irish comprised 85% of the total.

In each of the classes the various nationalities were represented in the same proportion as in the entire group, except insofar as this was influenced by the occupation.

Of the entire group	10.1%	Graded B
Of all native born	9.2%	Graded B
Of all the Italians	10.9%	Graded B
Of all the Irish	11.5%	Graded B
Of the entire group	7.1%	Graded C and D
Of all native born	6.1%	Graded C and D
Of all the Italians	9.7%	Graded C and D
Of all the Irish	5.0%	Graded C and D

Table 2. Grades

RACE:

	Class A	Class B	Class C	Class D	Total
White	770	94	37	23	924
Negro	58	7	8	3	76
	828	101	45	26	1000

Table 3

Of the negro race, 14.3% were in the C and D classes, while among the white, 6.5% were in these classes.

The number examined was too small to permit of any conclusions.

AGE:

Average age of the entire group	35.36 years
Average age of Class A	33.3 years
Average age of Class B	44.4 years
Average age of Class C	45.4 years
Average age of Class D	45.7 years

Age as a factor in the rating is demonstrated by the decreasing percentage of Class A in the older age group. The curve is steadily downward. The

greatest drop was in between the ages of 37 to 50, and this group constituted half of those examined.

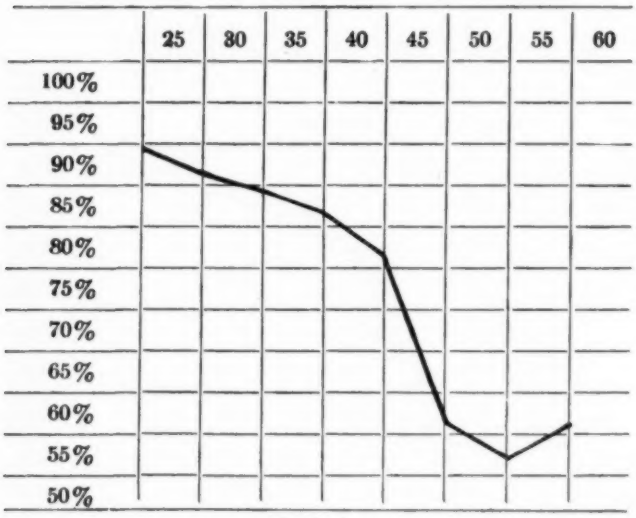

Table 4

Age in spans of five years	Class A	Class B	Class C	Class D	Total
20-25	75	3	1		79
26-30	134	7	2		143
31-35	155	12	6	1	174
36-40	192	14	6	4	216
41-45	138	15	8	5	166
46-50	72	25	8	13	118
51-55	39	15	10	2	66
56-60	23	10	4	1	38

Table 5. Age Groups

OCCUPATION:

Drillers and blasters formed a special occupational group as opposed to others:

Of the drillers and blasters, 26, or 13.7%, Graded C and D.

Of all the others, 45, or 5.5%, Graded C and D.

This disproportion is ascribed mainly to the occupation.

	Class A	Class B	Class C	Class D	Total
Laborers	329	33	11	9	382
Timbermen	175	20	12	1	208
Drillers	132	7	11	11	161
Blasters	25	2		4	31
Engineers	62	11	3		76
Nippers	8				8
Pipefitters	17	5	2		24
Metal lathers	11	2	1		14
Machinists	15	1	1		17
Signal men	8				8
Chauffeurs	5				5
Watchmen	26	20	4		50
Foremen	15			1	16
	828	101	45	26	1000

Table 6. Occupations

INDUSTRIAL HISTORY—solely as to dust exposure:

Persistent questioning of the applicant was the sole source of this information and related to his prior work in tunnelling, drilling, mining, foundries, blasting, stone cutting and with asbestos. Frequently the same individual was exposed to more than one of these hazards.

There was no history of exposure in 506 men.

Of the 494 men exposed, 468 showed no demonstrable pulmonary damage. The average exposure incurred per man was 8.4 years. Of the remainder, 26 showed silicosis, and these constituted Class D.

The following table (7) as to duration and source of the exposure is compiled entirely on the applicant's statement. It is largely correct, but not so in every instance.

The type and length of exposure of each individual case comprising Class D (Silicotics) is shown in Table 7.

The listed occupation is the one most consistently pursued.

The average exposure of each member of Class D was 17.6 years, exclusive of exposure incidental to ordinary laboring work engaged in at intervals by a good many.

Case No.	Foundry	Tunnel	Mining	Drilling	Stone-cutting	Blasting	Total years exposure
1. laborer		7	3		2		12
2. driller		1		3			4
3. laborer			2				2
4. driller				28			28
5. driller		9	1	7		8	25
6. blaster		8				4	12
7. laborer		2	4				6
8. driller		5		23			28
9. laborer	9	20					29
10. driller			13	3			16
11. blaster		10				24	34
12. driller		4	4	15			23
13. driller			1	15			16
14. driller		33			3		36
15. blaster		13		4		1	18
16. driller		19		3			22
17. driller		2		22			24
18. laborer			7				7
19. laborer		10					10
20. timberman		3					3
21. laborer		8					8
22. driller		6		10	3		19
23. laborer			28				28
24. driller	1		2	17			20
25. blaster						14	14
26. laborer		5					5

Table 7. Occupations, Class D, Silicotics

PREVIOUS EXAMINATIONS:

Antecedent pre-employment examinations elsewhere had been made in 566. On this examination, 36 of that number were found totally unsuited for the work contemplated.

Table 8 records the interval between the examinations:

Years	1	2	3	4	5	6	7	10
Class C	11	7			1		1	3
Class D	7	2		1				3

Table 8.

FAMILY HISTORY AND PRESENT COMPLAINTS:

This information, for obvious reasons, is highly inaccurate and is, therefore, omitted. It is only of passing interest to note that of the 1000 men examined, 27 complained of occasional colds, six of frequent colds, 13 of occasional cough, seven of occasional epistaxis, 12 of occasional sore throat, six of shortness of breath, one of pleurisy, four of lumbago, four of constipation, one of nervousness and two of frequent headaches.

In two, there was a family history of tuberculosis. In one instance, a history of previous tubercular infection was given. No one had any serious complaints at the time of the examination.

HISTORY OF PRECEDING DEFECTS AND DISEASES:

Under this caption is included the record of antecedent surgical procedures, various injuries, acute infectious diseases, constitutional conditions, etc.

Surgical Procedures:

Appendectomy	63
Cholecystectomy	2
Cyst removal	2
Eye—cataract removal	1
Hemorrhoidectomy	3
Herniotomy	41
Hydrocele operation	1
Liver abscess	1
Mastoidectomy	6
Nasal operation	7
Nephrectomy	3
Rectal abscess and fistulae operation	3
Semilunar cartilage removal	4
Tenoplasty	1
Testicle—removal	2
Thyroidectomy	1
Tonsillectomy	37
Tumor removal	2
Varicocele operation	1
Varicose veins	1
Wry neck	1

Traumatic Conditions:

Amputation	35
Fingers 29	
Toes 6	
Burns	3
Dislocations	1
Shoulder 1	
Fractures	58
Gunshot wounds	7
Stab wounds	1

Acute Infectious Diseases:

Bronchitis	5
Diphtheria	13
Erysipelas	3
Influenza	16
Malaria	11
Pleurisy	8
Pneumonia	29
Pulmonary tuberculosis	1
Quinsy sore throat	2
Rheumatic conditions	7
Scarlet fever	18
Smallpox	1
Typhoid	9
Whooping cough	44

Constitutional and Glandular:

Asthma and hay fever	7
Diabetes	1
Gastric ulcer	1

Venereal Diseases:

Gonorrhea	35
Syphilis	3

Miscellaneous:

Arthritis	1
Lumbago	1
Otitis media	1
Sciatica	2

Examinations:

WEIGHT—A limited variation from the figures in commonly accepted weight tables is of no significance. Therefore, we have allowed a margin of 10% each way in those termed arbitrarily of normal weight.

	Entire Group	Class A	Class B	Class C	Class D
Normal	605	508	57	23	17
Underweight	157	118	20	14	5
Overweight	238	202	24	8	4
	1000	828	101	45	26

Table 9. Weight

Variations above or below the "normal" were not consequential unless pronounced or associated with other abnormalities. The two tables directly following show to what extent such deviations prevailed.

	Class A	Class B	Class C	Class D
Minus 10% to 14%	70	9	5	3
Minus 15% to 19%	32	6	5	1
Minus 20% and more	16	5	4	1

Table 10. Underweight

	Class A	Class B	Class C	Class D
Plus 10% to 14%	80	10	2	0
Plus 15% to 19%	46	4	3	1
Plus 20% and more	76	10	5	3

Table 11. Overweight

There have been no significant weight changes in the past five years.

VISUAL ACUITY—Tested for distant vision:

Basis of rating:

"Average—20/20 to 20/40 inclusive.

"Fair"—20/50 to 20/65 inclusive.

"Poor"—20/100 and less.

Where one eye was "average" and the other "fair"—rated at "average."

Where one eye was "average" and the other "poor"—rated at "fair."

Unless there was complete loss of vision in the defective eye—rated at "poor."

Where one eye was "fair" and the other "poor"—rated at "poor."

	Entire Group	Class A	Class B	Class C	Class D
Average	935	791	86	37	21
Fair	50	33	8	6	3
Poor	15	4	7	2	2

Table 12. Eyes

The defective vision was uncorrected and in six cases constituted a serious impairment.

BLOOD PRESSURE:

The standard or normal pressure was fixed at 100 plus the age. As the readings in many instances were affected by factors extraneous to the physical condition, such as excitement, apprehension, etc., a variation of 10 millimeters above and below this fixed figure was included in the normal.

	Entire Group	Class A	Class B	Class C	Class D
Normal	686	587	62	27	10
Increased*	38	20	10	8	
Decreased	276	221	29	10	16
	1000	828	101	45	26

* Of this number, in only 20 cases was the increase great enough to be considered pathological.

Table 13. Blood Pressure

TEETH:

These are rated as "good," "fair" or "poor." Where the teeth were entirely, or in the main artificial, they were rated as "poor."

	Entire Group	Class A	Class B	Class C	Class D
Good	398	352	30	12	4
Fair	280	233	25	12	10
Poor	322	243	46	21	12
	1000	828	101	45	26

Table 14. Teeth

X-RAY FINDINGS:

Significant x-ray findings are shown in Table 15.

	Class A	Class B	Class C	Class D
Silicosis*				26
Tuberculosis			21	
Healed tuberculosis	4	14	4	
Abscess			1	
Periodical infiltration—marked	4	2		
Bronchiectasis		1		
Excessive hilar thickening	1			
Pleural thickening	10			
Enlargement of heart	24	8	7	
Aortic dilatation	7	3	2	
Aortic aneurysm			2	
Enlarged thyroid	1			
Substernal thyroid	1			
Cervical ribs	2			

* Includes cases with infection.

Table 15.

Aside from the silicosis and tuberculosis, the findings in main were similar to those usually encountered, and are recorded as of general interest.

ABNORMALITIES and DISEASES:

All of these merit consideration. Some, however, are particularly important in that they directly affected either work capacity, were progressive or a source of danger in the event of injuries. These are set forth in capitals:

Cardiovascular System:

Heart—
- Enlargement — 18
- Myocardial degeneration — 22
- VALVULAR DISEASE — 8

Blood Vessels—
- ANEURYSM — 2
- AORTITIS — 7
- ARTERIOSCLEROSIS and HYPERTENSION — 20
- Varicose veins — 115
  - Small — 74
  - MEDIUM — 26
  - LARGE — 15
- Hemorrhoids — 75
- Varicoceles — 19
  - Small — 15
  - LARGE — 4

Respiratory System:

Upper—

Nose
- Obstruction — 116
  - Deviated septum
  - Hypertrophied turbinates
- Perforated Septum — 2

Throat
- Diseased tonsils — 85
- Pharyngitis — 79

Asthma — 1

Bronchiectasis — 1

Bronchitis — 16

Lower
- PULMONARY TUBERCULOSIS — 30
- Arrested tuberculosis — 22
- LUNG ABSCESS — 1
- SILICOSIS — 26

Digestive System:

Cirrhosis of Liver — 1

Hemorrhoids (See blood vessels)

Genito-Urinary System:

Kidneys
- NEPHRITIS — 3

Genitalia:

Testicles—
- Atrophied — 6
- Undescended — 9
- Epididymitis — 1
- Hydrocele — 4
- Varicocele (See blood vessels)

Phimosis — 1

Venereal
- GONORRHEA — 1
- SYPHILIS — 4
  - Early — 2
  - Late — 2

Herniae
- INCISIONAL — 3
- INGUINAL — 27
  - Unilateral — 21
  - Bilateral — 6
- Umbilical — 14
- VENTRAL — 1

Skin Conditions:

Acne (pronounced) — 15

Dermatitis — 5

Eczema — 5

Fibroma — 1

Hemangioma — 1

Herpes zoster — 1

Lipoma — 8

Moles (multiple pigmented) — 1

Psoriasis — 2

Tinea — 3

Glandular and Constitutional:

DIABETES — 6

Thyroid enlargement — 3

Malnutrition — 3

Skeletal—Muscular and Nervous System:

Ganglion — 1

Spinal curvature	15
MARKED IMPAIRMENT OF EXTREMITY	7
Flat feet	126
first degree	84
second degree	35
third degree	7
TABES DORSALIS (See Genito-urinary system)	
Special Senses:	
Eyes	
CATARACT	2
Corneal opacity	5
Conjunctivitis	15
Exophthalmos	1
Fixed and irregular pupils	9
Strabismus	3
VISION—marked defect	6
Ears	
Defective hearing	75
Otitis media	4

Specific causes for "B" classification (Those subnormal, due to the presence of one or more conditions, which either of themselves or because of their potentiality make their employment an undue hazard).

Frequently there were other contributing causes—but only the main condition is listed.

Cardiac condition (myocarditis and valvular disease)	1
Cirrhosis of liver	1
Defective vision—marked	6
Epididymitis—acute	1
Hernia	30
Hypertension—including arteriosclerosis	17
Marked impairment of limb	7
Nephritis	2
Pulmonary tuberculosis—arrested	14
Subnormal general condition	3
Syphilis	1
Tabes dorsalis	1
Upper respiratory infection—acute	1
Varicose veins	7
Total	101

Specific causes for "C" classification (Those having one or more major conditions which made exposure incidental to this work inadvisable).

Aneurysm	2
Cardiac condition (myocarditis and valvular disease)	5
Diabetes mellitus	6
Hernia (huge ventral)	1
Hypertension with arteriosclerosis—complicated	3
Lung abscess	1
Nephritis—complicated	1
Pulmonary tuberculosis	21
Syphilis—active	1
Tabes dorsalis—advanced	1
Tabes dorsalis—advanced	1
Varicose veins—extensive	3
Total	45

SILICOSIS—CLASS "D":	
First stage	7
Second stage	6
Third stage	4
With infection*	9
Total	26

(* Tentative diagnosis made mainly upon x-ray films. There were no sputa examinations).

Recapitulation of data appertaining to this class:
White .....23; Negro 3

Average age, 46.7 years. The youngest was 31 years of age. Half of them were between 46 and 50 years, and the oldest was 60 years of age.

The average exposure in dust hazard occupations was 17.6 years.

The complaints were meager, consisting of occasional cold in five, intermittent cough in three, shortness of breath in two, and all had been in continued employment the preceding five years.

As in the general experience, the diagnosis could not have been made without the x-ray. physical signs were inconclusive, and in 10 cases there were no physical signs in the chest.

COMPARISON with analogous recent examinations totaling several thousands in other industries:

Construction Company.....designated Series I (subject matter of this digest)
Chain Stores.....designated Series II
Milk Distributing Co.....designated Series III

Average age:

Series I	35.4 years.
Series II	25.6 years.
Series III	30.6 years.

A comparative study of the weights in these Series showed that a much larger percentage was underweight in Series I. This is probably explained by the lower age and also by the fact that, because of their physique, employment of a less laborious nature was chosen.

Uncorrected errors of vision:

Series I	3.5%
Series II	5.9%
Series III	7.0%

Hernia:

Series I	3.1%
Series II	2.5%
Series III	4.8%

Comment: There was approximately the same percentage of antecedent hernia operation in all three Series (4.0%).

Varicose Veins:

Series I	11.5%
Series II	4.8%
Series III	6.0%

Applying similar standards of rating to the members of all three "Series" it was noted that those below the average in:

Series I were	17.2%
Series II were	7.0%
Series III were	9.0%

The much higher percentage in Series I may be due in part or wholely to the following;

1. Greater age;
2. The character of the occupation and its concomitant hazards;
3. Habits and environment;
4. More extensive examination which included x-ray of the chest.

It is evident from the foregoing data that:

The strenuous vocations of the older age group are associated with greater incidence of diseases.

The frequency and severity of the diseases encountered warrants their prompt consideration.

Their recognition seemingly has only been commonly achieved by the media of routine pre-employment examination.

Ascertaining their presence is the initial step towards:

Correction;

Prevention of further retrogression, or finally, when indicated,

A change of occupation.

Carbon Tetrachloride Poisoning

—Outline of a Successful Treatment—

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A RECENT case of carbon tetrachloride poisoning has stimulated our interest in the clinico-pathological aspects of this industrial hazard, and, utilizing the clinico-pathological background as a rationale for treatment, we herewith submit an outline of therapy for carbon tetrachloride poisoning which has been successful in our hands.

The ever increasing literature on this subject reveals the enormous amount of experimental and clinical work that is being done all over the world to alleviate the pessimistic prognosis of this poisoning, and forms the foundation of this discussion.

Report of a Case

THE patient, a white male, 47 years old, employed as a cable-splicer for six months, was admitted to the hospital on June 4, 1936, at 11:10 A.M., with the following complaints: headache, nausea, vomiting, and inability to urinate.

ONSET AND COURSE: The patient stated that he was apparently well up to May 31, 1936, when, while working in a manhole, 15 ft. x 12 ft. x 12 ft., with four other men, making a "pothead", and cleaning insulators with a mixture of gasoline and carbon tetrachloride, he noticed that the manhole became filled with the fumes of the tetrachloride mixture to which he was exposed about four and one-half hours. He stated that after about three hours in the pit he noticed a slight headache which became progressively worse, that soon after leaving the manhole he grew dizzy and nauseated, and vomited, and that he had since been unable to retain anything in his stomach. Upon arrival at home his symptoms increased in intensity and he noticed a scantiness in urination, increased severity of the headache, and a pain in both lumbar regions. He said that his urine was very dark shortly after he came out of the manhole. He stated that he worked the day after his exposure to the fumes, but felt sluggish and dizzy. He continued throughout the day, however, haphazardly doing his work, and on June 2 he noticed an increase in the pain in his back, and the appearance of pain in the calves of his legs. He did not recall any convulsive seizures. He mentioned the interesting fact that his weight increased from 174 pounds on May 30, to 180 pounds on June 4, 1936.

He reported that the other men working in the manhole felt the effects of the fumes for few days, namely, dizziness, and headache, and explained that the mixture contained two parts of carbon tetrachloride to one part of gasoline.

PHYSICAL EXAMINATION revealed a moderately developed white male, 47 years of age, having a pasty complexion, and complaining of anuria and dizziness; temperature 97.8°F; pulse, 80; respiration, 28; blood pressure, 170/90.

The general physical examination was essentially negative, the heart and lungs were normal, the liver and spleen were not palpable, and the lumbar regions were moderately tender but the kidneys were not palpable.

The patient was apprehensive and restless. He was put on a liquid diet and catheterized without obtaining any urine. Intravenous fluids were immediately administered, and he was given 750 cc. of normal saline solution and 1000 cc. of saline glucose mixture. Throughout the day of his admission the total fluid intake by mouth and intravenous fluids was 3500 cc. of fluid, with an emesis of 600 cc. He voided only 3½ ounces of urine. He became progressively restless, complaining of a severe headache and pain in the precordium. He was given 15 grains of calcium gluconate intravenously every four hours, and the saline glucose mixture continued.

LABORATORY FINDINGS ON ADMISSION: Red blood corpuscles, 3,720,000; white blood corpuscles, 7,000, normal differential.

Blood chemistry, non-protein nitrogen 150 mg. per 100 cc. blood; glucose, 55 mg. per 100 cc. blood.

First urine specimen contained 4-plus albumin, few hyalin casts, many erythrocytes, many leucocytes, cylindroids, and amorphous phosphates.

DIAGNOSIS: Carbon tetrachloride poisoning, nephritis with anuria, and impending toxic uremia.

CLINICAL COURSE: Realizing that the therapeutic armamentarium available was vast, it was only a question as to what procedure to follow, and to do the right thing at the right time. We shall, therefore, list the therapeutic routine as the symptoms develop, and try to evaluate the rationale of these measures as we progress.

The patient's anuria persisted despite his enormous intake of fluid and, associated with the anuria, he became dyspneic and cyanotic, and complained of increasing precordial distress. His blood chemistry obviously had to be rectified, and in view of the hypoglycemia and increasing non-protein constituents of the blood, associated with a loss of chlorides due to the persistent emesis, an electrolyte disbalance was present with an impending acidosis. Therefore, the best possible method available for correcting these conditions would be the furnishing of electrolytes by means of Hartman's Buffer solution, a modified Ringer's solution, alternated with intravenous infusion of 25% glucose, 250 cc. to 500 cc., which would serve to bank up the liver and, due to its hypertonicity, serve as a diuretic. Small doses of insulin were given, five to 10 units t.i.d. to facilitate the oxidation of the intermediate products of protein, fat and carbohydrate metabolism.

For the precordial distress, theobromine sodium salycilate, grains 60 per rectum, with starch, was given, with the hope that the zanthane effect would relieve the coronary embarrassment, and at the same time assist with the diuresis.

Though these measures relieved the patient, symptomatically, his anuria persisted despite the fact that he was taking 3000-5000 cc. of electrolytes, fluids and glucose, his urine output averaging 300 cc. daily for the first four hospital days, June 4 to 8. He was given added glucose in the form of Karo syrup by mouth with the intention of conserving the integration of the liver and at the same time to relieve the load of the intravenous therapy on the veins. Karo syrup was taken moderately well, two ounces q.i.d. being given, which the patient tolerated. On the fifth hospital day he became markedly distended. There was a marked expulsion of flatus after a S.S. enema, and he was more comfortable. Cyanosis and dyspnea were still present but to a lessened degree, and the heart tones were of good quality and no apparent arrythmia was present.

The same therapy was continued without any remarkable change in the urine output until the eighth day of hospitalization when he had an intake of 3150 cc. by all routes, and voided 1420 cc. of dark urine. This was the first indication that our heretofore diuretic therapy had any effect of stimulating the secretion of urine. At this point, a relatively new drug was used which has proved successful in diseases of the peripheral-vascular system and, in view of the fact that associated with the toxic nephritis of the kidneys there is a concommitant spasm of the arterioles, it was thought best at this time to use an anti-spasmodic with the hope that this spasm might be relieved or a collateral circulation established. The drug we refer to is papaverin, which, when given in half-grain doses intravenously or by mouth, releases the vascular spasm both centrally and peripherally, and in our case, the release of the coronary symptoms and spastic state of the arterioles of the kidneys was imperative if an arrest of a terminal uremia was to be accomplished.

The drug did not fail us, and it is our belief that we have here a valuable aid in our armamentarium in combating anuria as associated with spastic states of the tubular blood vessels. The rationale for the use of this drug will be discussed under the pathology of carbon tetrachloride poisoning.

Subsequent to the use of papaverin, intravenously, the patient began to void, and the clinical record reveals the following results:

Hospital Day	Intake	Output
9th	3010 cc	2700 cc.
10th	4210 cc	3420 cc.
11th	3640 cc	3925 cc.
12th	3295 cc	3130 cc.
13th	3640 cc	3910 cc.
14th	3740 cc	4310 cc.
15th	3110 cc	3260 cc.
16th	2300 cc	2150 cc.
17th	1500 cc	2030 cc.

In order to augment the diuretic effect, sodium decholin was given, and none of the mercurial diuretics was used because of the existing nephritis which made them contra-indicated. The patient showed continuous improvement after secretion of urine was established, and the subsequent laboratory findings showed a decrease in the organic constituents with also a decrease in the albumin and red and white blood cells in the urine. The blood pressure returned to apparent normal levels, with an average blood pressure of 150/85. The Taka-Ara test for liver dysfunction showed flocculation in four tubes, pathognomonic of liver damage. The N.P.N. was 50 mg. per 100 cc. of blood; the urea, 25 mg. per 100 cc. of blood. The blood sugar was normal at this examination.

The Mosenthal test showed a moderately low fixed specific gravity ranging from 1.007 to 1.011, indicating residual, glomerular, and tubular pathology.

A recent Taka-Ara, Rosenthal, and Addis cell count of the urine revealed normal function of the liver and kidneys.

The patient improved gradually and was discharged from the hospital August 15, after 72 days of hospitalization.

Etiology and Pathology

CARBON tetrachloride finds rather wide uses in industry and the arts as a non-inflammable solvent for greases, fat and rubber, as a constituent of certain paints, as an agent for extinguishing fires (pyrene), and medicinally as a delousing agent, and an anthelmintic (especially against hookworm infections). Chemically, carbon tetrachloride is a heavy colorless fluid with an odor resembling that of chloroform. Its specific gravity is 1.584 at 25°C and it boils at 76.1 to 76.2°C. It is almost insoluble in water (1:1250) and glycerine, but mixes in all proportions with alcohol, chloroform, and oils. The fumes are very heavy, being 5.3 times as heavy as atmospheric air.

Moller[1] lists the following uses of carbon tetrachloride:

1. For removal of grease from metal parts. A brief washing with carbon tetrachloride removes grease and dirt better than most other cleaners. The fluid used for this work may be filtered afterwards, or, as is the practice in larger shops, it can be distilled and then used again. It is used in printing plants, and for cleaning boilers and the motors of automobiles and aeroplanes, for which it is particularly serviceable as it is non-inflammable.

2. For removal of grease in dry cleaning. In modern plants the substance is used in tightly closed apparatus which quite prevents the workers in the plant from becoming exposed to the fumes. Further, carbon tetrachloride is used to a large extent in many homes for the removal of grease spots.

3. In technology for the extraction of animal fats from various kinds of plant and animal substances, for example, in the pharmaceutical and chemical industries.

4. As a solvent for crude rubber, yielding a very sticky and non-inflammable solution of rubber.

5. In the vulcanization of caoutchouc, as a substitute for the volatile and very poisonous carbon bisulphide, where its employment has meant a great advance, both technically and hygienically.

6. As a solvent for lac, resin, etc.

7. In some forms of fire extinguishers carbon tetrachloride is used principally for extinguishing small fires that are due to ignition of benzine, benzol, gasoline, petroleum, etc. Fire extinguishers using carbon tetrachloride are, therefore, a very important part of the equipment in automobiles, aeroplanes, and motor boats. They are not serviceable in fires with a large supply of calories, and especially not in fires where there are large amounts of redhot iron.

8. As an anthelmintic drug carbon tetrachloride may be indisputably considered to be a specific remedy against hookworm intestinal parasites that are found all over the world. It may be given to entire populations with the assurance that it will remove from 95 to 100% of the hookworms harbored by infected individuals. The treatment is carried out in the following way: in the morning, after a light meal, the patient is given orally 2.5 to 3 cc. of the drug, whether in gelatine capsules or simply diluted with water, and three hours later a dose of magnesium sulphate. For children under 15 years of age the dose is 0.2 cc. per year of age. The treatment is practically without any inconvenience to the patient. Chronic alcoholism is a strong contra-indication. If the treatment fails, it must not be repeated within three weeks.

Moller[2] emphasizes against the use of carbon tetrachloride in beauty establishments, stating that its use is indefensible.

Jaksch[3] reports the sudden death of a woman in a London hair dressing parlor while her hair was being treated with a carbon tetrachloride solution.

McGuire[4] reviews the work done at the research laboratory of the Bureau of Mines, which demonstrated that when fire extinguishers (carbon tetrachloride) are used in a closed room, subject to high temperatures such as would occur in a fire, deadly gases are formed. Phosgene gas, which is very poisonous, is formed in toxic quantities, chlorine in smaller amounts; hydrogen chloride also is formed and there is considerable vapor of carbon tetrachloride. The experiment showed that it is extremely hazardous to employ carbon tetrachloride fire extinguishers in a fire in closed places.

Kionka[5] re-emphasized the necessity of precaution, and showed in experimental work on rabbits that if the rabbit is slightly burned, the carbon tetrachloride is absorbed much faster through the burnt skin, and at autopsy lesions compatible with those found in carbon tetrachloride poisoning in the human are found in the liver, spleen, and kidneys.

Individuals differ in their resistance to carbon tetrachloride. Obesity, use of alcohol, starvation, and in general, a poorly nourished condition seem to lessen the resistance and increase the toxicity of the blood. Malnutrition and calcium deficient diets, as shown by Minot[6] and Cutler[7], predispose to the severe effects of the carbon tetrachloride poisoning. In experiments on dogs, Cutler found that none of the diets used protected the liver from damage by carbon tetrachloride, but certain diets did definitely reduce the severity of the symptoms and the number of deaths secondary to the liver damage. Such a protective diet should

(a) not tend to increase guanidine as meat has been shown to do;

(b) be rich in calcium to combat guanidine; and

(c) be rich in carbohydrates to relieve hypoglycemia as it develops.

Gautier, et al[8], in an exhaustive pathological study of a case, conclude that accidental carbon tetrachloride poisoning with injury of the liver and kidneys and marked changes in the renal amino-genetic function may be followed by progressive alkalipenia, complicated chloropenia, and hyperazotemia.

Smyth[9] believes that the general impression as to the dangerous character of carbon tetrachloride as used in industry has little factual basis. He has been able to find in the literature and through interviews with experienced men, only 27 cases of acute poisoning, and of these, 14 followed its internal administration as an anthelmintic. Of the other 13 cases, only six occurred in the United States, and one of these was a baby who died after having adhesive tape strips removed with the help of carbon tetrachloride. He has found about 96 cases of acute and sub-acute non-fatal poisonings, but 18 of these were not industrial. Of the industrial cases, 33 occurred in this country, but not all of these are beyond doubt, since 13 were in persons using mixed solvents. Seven cases of chronic poisoning in industry were all that could be found, six were in this country. Interviews with authorities connected with the State labor departments and insurance officials bear out the author's contention that there is very little poisoning in proportion to the large amount used, which has increased from 11,745,972 pounds in 1923, to 34,095,302 pounds in 1931. The cases reported were from exposure to fumes in a close, poorly ventilated place, or to careless use, or to previous ill health, or to alcoholism, for, as has been shown experimentally, alcohol and either a calcium-poor or fat-rich diet will increase the severity of carbon tetrachloride intoxication.

Takahashi[10], in an exhaustive study of the metabolic relations in carbon tetrachloride poisoning, showed that there was an increase in the protein metabolism and, consequently, the intermediary products of metabolism, the sulphuric acid, phosphoric acid, and oxalic acid, beta-oxybutyric acid, hypuric acid, etc., were increased, and that abnormal acid formation leads to a condition of acidosis, and the increased excretion of ammonium nitrogen. Urea is the main nitrogenous end product of metabolism in the mammal. Its excretion increases with the increase of the total nitrogen; the urea excretion is as a rule increased in diseases accompanied by an increase in decomposition of the tissue proteins. The author has attempted to determine whether the increased excretion of ammonium in carbon tetrachloride poisoning is actually due to an increased acid formation in the organism. It was found that the acids formed in the organism can be neutralized by the introduction of alkali as a result of which the formation of ammonia becomes limited, which fact is utilized therapeutically in the use of Hartman's Buffer solution.

Pathologically, Takahashi's experimental animals exhibited the following findings: the livers appeared yellowish white, and somewhat cloudy, and in some places the lobular striations were indefinite. The liver substance was of doughy consistency; the kidneys, heart, lung, and spleen showed no macroscopic changes.

Microscopically, the protoplasm of the liver cells was fine, or coarsely granular, and the nuclei showed some mitosis. The liver cells presented a picture of hypertrophic compensation. The Kuppfer stellate cells and hemosiderin containing cells were seen here and there. Around the central vein there were seen some degenerating cells. Along the vascular walls there was present some cellular infiltration. Glisson's capsule was well developed. Here and there, capillary hemorrhages were present, some capillary hemorrhages were found in the kidneys, especially in the medulla. The

glomeruli were swollen and some exudate was present in Bowman's capsule. Fatty granules were seen in Henle's loops. The lungs were hyperemic, atelectatic in some places, and the peripheral small bronchii contained some hemorrhagic fluid. The follicles of the spleen were slightly atrophic, there were present hemosiderin deposits, in the pulp there were present hemosiderin containing cells. Areas of necrosis were present in the cortex of the suprarenals.

Poindexter and Greene[11] reported a case of toxic cirrhosis of the liver due to carbon tetrachloride poisoning showing infarcts of the right kidney with compensatory hypertrophy of the left kidney. Their case was one of long standing exposure which resulted clinically in cirrhotic syndrome with ascites.

Dudley[12] reported a case of toxic nephritis with uremic manifestations.

Mauro[13] pathologically demonstrated fatty degeneration of the liver, fat drops in the epithelial cells of Henle's loops, and in the convoluted tubules, also alterations in the suprarenals, in the form of necrosis were encountered in the cortical cells. The author states the toxic substance probably acts on the cells of the renal epithelial which line the convoluted tubules and Henle's loops. In consequence, they swell and vacuolize, and in grave cases break up and fall into the lumen of the canaliculum and cause an obstruction which may result in complete occlusion. This may explain oliguria or anuria, which is encountered in such cases. The vascular tufts show diapedesis. Generally, when the patient is treated in time, a rapid regeneration occurs, after the epithelial cells have been thrown off.

Mohamed[14] states that the liver shows larger areas of local necrosis around the hepatic lobules. The necrosed areas occur in the form of triangular or irregular zones communicating with each other by thin strands of necrotic liver cells. There is an infiltration of round cells in these necrosed areas and in the portal tracts, also a certain amount of fibrosis, showing an early process of fine cirrhosis. In some of the necrotic zones, especially near the surface of the organ, the sinusoids around the dilated central veins are seen markedly congested. In the remainder of the necrotic areas, the sinusoids are empty. The kidneys show that the majority of the convoluted tubules have undergone necrosis of their columnar epithelium. Only a few tubules have escaped the change and in the lumen of these are seen the desquamated epithelial cells.

Some of the tubules contain blood, and there is patchy congestion of the inter-tubular capillaries. In the medulla, a large number of the collecting tubules show necrosis of their lining epithelium and some of them contain fresh hemorrhage. This necrosis is less severe than in the convoluted tubules, because the cells have their nucleii still present although the protoplasm has broken down into a granular detritus. The intact tubules contain brown pigment in some of them. Some of the vessels here also show marked congestion, and the fibrous tissue framework is also increased.

The aforementioned pathological lesions of the kidney, from a physiological point of view, would suggest associated spasm of the arterioles as demonstrated by diapedisis, exudation, and necrosis, which conditions we think may be kept from progressing by the use of an anti-spasmodic such as papaverine hydrochloride or sulphate.

Symptoms

THE symptoms are the clinical manifestations of the pathological changes occurring in the various organs in the body associated with altered metabolism and disturbed tissue and blood chemistry. Depending of course, upon the individual's susceptibility, nutritive state, inebriation from alcohol, and intake of calcium. The onset is usually insidious, but may be acute.

Davis[15], from a number of experiments and examinations of many persons exposed to carbon tetrachloride fumes, enumerates the following possible symptoms: slight headache; nausea—in many cases this may become severe and last for several days; nervousness; mental confusion; loss of weight; dry dermatitis; secondary anemia; jaundice; slight to severe chronic spasm of the muscles; necrosis of the liver — manifested by jaundice; acites; coma; acidosis; phosphaturia and irritative nephritis; loss of consciousness; coma and death, and visual disturbances — such as blurred vision, color confusion, and disturbance of near vision.

The author states that dilutions of 0.01% show very little effect over a period of from six to eight hours, but if continued daily without intermediate aeration, accumulative effects are shown and chronic tetrachloride poisoning occurs. Concentrations of 0.1 to 0.5% produce very little effect if only a short exposure is made and not repeated until the next day. He states that the principal avenue of absorption is the respiratory tract and that individuals with the respiratory diseases and with impaired myocardiums are definitely more susceptible.

Wirtschafter[16] describes five cases of carbon tetrachloride intoxication in men employed in dry cleaning and exposed to fumes eight to 10 hours daily for one to six months. All complained of headache, dizziness, nausea, vomiting, diarrhea, with abdominal pain associated with weakness and loss of weight, insomnia in one, irritability in another, and lethargy in a third. One man had polyuria; three of the men complained of visual disturbances, spots before the eyes, blurring, "everything looked small"; and all, including the two who had no such symptoms, showed bilateral, peripheral constriction of the color fields, but no central scotomata. Typical toxic amblyopia is characterized according to Knapp, who takes carbon disulphide as the typical poison by a central defect with normal periphery. The symptoms, dimness of vision with alteration of the color fields, are the same as those in the author's case.

Acting on Lamson's[17] discovery in animal experiments that carbon tetrachloride produces necrosis of the liver with a subsequent fall in the blood sugar, Wirtschafter made blood sugar tolerance analyses and found the amount in all five men at the lower border of the normal variation, and in some analyses below this; while in the urine there were slight amounts after the administration of glucose. The blood sugars, the urines, and the visual fields returned to normal after the men left their work and were put on a high calcium and dextrose diet. The explanation for the symptoms and the success of this treatment the author finds in Adler's [18] recent book on the eye.

Adler has demonstrated that that portion of the vitreous humor which is in close contact with the retina contains less sugar than the more anterior layers, and that this is due to the greater glycolytic power of the retina. The latter utilizes rapidly the available glucose and leaves the vitreous with a low sugar content, all sugar entering the vitreous having to pass first through the retinal circulation. He suggests that "possibly the retina not having storage facilities for glycogen is forced to use the glucose circulating in the blood." Wirtschafter thinks that as a result of the low blood sugar caused by carbon tetrachloride intoxication the retina is deprived of an adequate supply of glucose for normal metabolism and function, inasmuch as there is also a deranged fat metabolism in such intoxication, and, according to Adler, some substance present in the liver and certain fats is concerned with the rod-visual purple function of the retina. He thinks the explanation for the alterations of color vision may be found there.

Hunter[19] says the early stages of the illness are characterized by persistent headache, nausea, vomiting, diarrhea, and tenderness over the liver, but such symptoms are often followed by oliguria, suppression of urine, and uremia. The death rate is low, and a person with jaundice, suppression of urine, and uremia, may completely recover.

Hamilton[20] states: "It is well to remember that human poisoning differs from experimental animal poisoning in some respects. In the latter, it is the damage to the liver that dominates the picture. The kidneys are much less involved. In man, although there is at first severe vomiting, intestinal pain, diarrhea, jaundice, and tenderness over the liver, and an excess of blood urea and diminished urine that may also be traced to liver damage, still, in fatal cases, there is evidence of profound involvement of the kidneys, increasing oliguria, generalized edema, finally complete anuria and convulsion."

Martin[21] observed hemiplegia with loss of reflexes, bilateral Babinski, subcomatose condition, slight conjugate deviation of the head and eyes, vomiting without nausea, and rapid pulse.

Lehnherr[22] reports his roentgenological studies of a case which revealed a diffuse bilateral mottling as seen in bronchopneumonia or advanced tuberculosis; it was thought to be due to multiple fat emboli in the lungs, which were observed by McMahon and Weiss[23] who, in their studies, were able to demonstrate macroscopic fat in the pulmonary artery.

The electro-cardiogram may show progressive changes, depending of course, upon the degree of the intoxication. Flattening of the T waves may occur especially in lead I with inversion of T 2 and T 3. However, more study along this line is necessary in order to reach any adequate conclusions; though the precordial distress seen in some cases is somewhat dependent upon the arteriosclerotic changes which are activated to progression by changes in the plasma liquids during carbon tetrachloride poisoning.

Laboratory Findings

THE blood count usually shows a secondary anemia at the onset with a more or less unchanged picture of the white corpuscles. The urine is usually highly colored at the onset with a high specific gravity and may contain visible bile and blood. The phenolsulphonphthalein test shows abnormal retention of dye, and the urea and creatinine show marked increases with a hypoglycemia due to liver involvement. There is a high icteric index, retention of the bromo-sulphaline dye, positive Taka-Ara reactions indicative of liver dysfunction. There may be also a disturbance of the ester cholosterol-total cholosterol ratio. All these factors return approximately to normal upon recovery, leaving evidences of only very little if any residual pathology, if proper therapy is instituted.

Treatment

THE prophylactic treatment is essentially to be cognizant of the various factors brought out by Moller,[1,2] Davis,[15] Dudley,[12] Hamilton,[20] Mauro,[13] etc., as to the contributing causes of carbon tetrachloride poisoning, and to make an effort to rectify those conditions which do not come up to the standards and specifications of various industrial hygienists as being safe for the worker in the plant using carbon tetrachloride.

Active Treatment

ACTIVE measures should be used as soon as the patient is observed complaining of the various bizarre symptoms subsequent to exposure to injurious quantities of carbon tetrachloride. Immediate removal or withdrawal of the poison is essential to successful treatment. If one bears in mind the complex clinico-pathological picture and disruption of the normal body chemistry, the treatment resolves itself into the following procedures:

1. Intravenous hypertonic glucose and Hartman's solution to combat acidosis, and liver dysfunction.
2. Insulin in small doses, usually two to three units of U 20 to facilitate carbohydrate fat and protein metabolism.
3. Calcium gluconate or calcium lactate as recommended by Minot[6] and Cutler[7].
4. Papaverine hydrochloride or sulphate, an alkaloid of opium, grains one-half intravenously where coronary, kidney, and spastic states are present.
5. Sodium decholin as a diuretic to facilitate the action of papaverin.
6. The zanthane diuretics to relieve the coronary embarrassment and to facilitate diuresis—however, we think the papaverine effect is slightly better.
7. Never use epinephrin, since the Jourdans[24] have observed syncope subsequent to its use in carbon tetrachloride poisoning.
8. Methylene blue may be used as recommended by Nelson,[25] but we have had no experience with this method.
9. Oxygen inhalation may be necessary if pneumonic symptoms are present.
10. The administration of a high carbohydrate diet is essential.
11. Blood transfusion may be necessary for anemia.

Summary and Conclusions

WE HAVE reported a case of a so-called mixed carbon tetrachloride poisoning in which the clinical picture of carbon tetrachloride poisoning predominated.

2. We have outlined a course of therapy based on physio-pathological studies.

3. The administration of glucose and available electrolytes is essential.

4. The use of insulin augments the effect of the electrolytes and glucose.

5. Anuria may be overcome by an important adjunct associated with the aforementioned therapy, namely, papaverin, a drug used to overcome spastic states of the blood vessels.

6. The wide use of carbon tetrachloride in industry makes it a rather formidable poison when individuals who are susceptible (Refs. 6 and 7) are exposed.

7. The clinico-pathological findings manifest disturbed metabolic states.

8. These metabolic states are successfully rectified with the aforementioned therapy.

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Occupational Diseases

—and the Illinois Law—

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I DEEPLY appreciate the honor of discussing the subject of occupational diseases and of the new Illinois Workmen's Occupational Diseases Act, at this meeting of the Central States Society of Industrial Medicine and Surgery and its distinguished members and friends.* I desire to congratulate this association upon its interest and activity in the field of occupational diseases, because it has just recently, and suddenly, become a matter of tremendous importance and concern to employers, employees, and the general public, and it is a field in which the medical profession, and particularly those concerned with industrial medicine and surgery, must play a most important part.

The general subject of occupational diseases, or the matter of a particular occupational diseases law, includes many separate yet closely related and, in fact, interdependent, subjects and problems, none of which could be adequately considered in a single discussion or article. A particular law, such as the new Illinois law, may present certain particular or peculiar problems, but essentially all problems related to the general subject are the same.

As I have stated, the general matter of occupational diseases and occupational disease legislation has recently and suddenly become a matter of tremendous importance and concern. As regards the new law, employers and employees, the medical and legal professions, all those concerned with the occupational diseases law, had little, if any, knowledge of occupational diseases generally, or of the Illinois law in particular until after it had been submitted to the legislature as an agreed bill, and had been enacted as law, without change or amendment. I consider that a knowledge of the nature and importance of the many problems involved, and of the need for immediate, intelligent action, is of paramount importance to a successful application and administration of the new law, and to a solution of the general problem, because, as in any case, a realization and understanding of the problem is perhaps the most important step on the way to its solution.

I shall, therefore, address my remarks to matters that will aid in understanding the nature and importance of the general problem, with some reference to the Illinois law; and briefly discuss:

The purpose of occupational disease legislation;

The problems and dangers in its application and administration;

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The history and background of the Illinois law; and

The extent of knowledge and precedent for such legislation and its administration.

And then I shall make some suggestions, particularly to the medical profession, but also to all those concerned with occupational diseases, as to action that should be immediately taken.

THE essential purpose of occupational diseases laws—as of the new Illinois Law—is to extend, to disability and death due to occupational diseases arising out of and in the course of the employment, the principle of compensation now generally provided by law in practically all states for disability and death due to accidental injuries arising out of and in the course of employment.

A number of states now have laws for such purpose. Some of them, including the Illinois law, are what are called general or all-inclusive of all occupational diseases. In the laws of other states, and in all foreign countries — including England, the continent, Germany, Canada, South Africa, and the British dominions, which have had such laws for years, with scientific medical study and investigation of industries and occupational diseases, and have had medical advisory or appeal boards—the schedule method of enumeration of specific occupational diseases, and including only those specifically named in the law, has been uniformly followed.

A schedule form law affords definite information to an employer as to the liability under which he operates, and as to the diseases or hazards as to which preventive measures must be used. It also informs the employee as to diseases in his employment and against which he should guard, and also as to causes for which he may be compensated in the event of disablement. A schedule form of law aids certainty of application, facilitates administration, and tends to lessen litigation. The general plan in such a law is to schedule such occupational diseases as have been definitely recognized as existing in industry, and as to which there is definite knowledge. Other occupational diseases may be added as they become known and understood. For illustration, shortly before the Illinois law was submitted to the legislature there was a great deal of publicity given to a few cases of radium-dial poisoning in one plant in Illinois. Radium dial poisoning had become definitely known in some states, and was definitely diagnosable, in 1925 or 1926, and in some states was specifically added to the schedule of occupational diseases. In Illinois any such employment could have been added to employments using lead, etc., under the old law; or, if a schedule-form new law had been enacted, such an occupational disease could have been specifically scheduled.

A general or all-inclusive law, on the other hand, is vague and uncertain; usually indicates a lack of legislative study or investigation by a commission or otherwise; places upon administrative bodies or commissions the burden of determining what may be occupational diseases under its provisions; and leaves employer and employee alike without essential information. The vagueness and uncertainty of such a law puzzles employers, employees, commissions and all having to do with the law, and necessarily increases litigation. The tendency will be for claims to be made for every sickness, disability and death, leaving certainty, precedent and understanding to develop after, perhaps, years of litigation.

The provisions of the Illinois law as to the occupational diseases covered by the law:

"SECTION 6. In this Act the term 'Occupational Disease' means a disease arising out of and in the course of the employment. Ordinary diseases of life to which the general public is exposed outside of the employment shall not be compensable, except where the said diseases follow as an incident of an occupational disease as defined.

"A disease shall be deemed to arise out of the employment, only if there is apparent to the rational mind upon consideration of all the circumstances, a direct causal connection between the conditions under which the work is performed and the occupational disease, and which can be seen to have followed as a natural incident of the work as a result of the exposure occasioned by the nature of the employment and which can be fairly traced to the employment as the proximate cause, and which does not come from a hazard to which workmen would have been equally exposed outside of the employment. The disease must be incidental to the character of the business and not independent of the relation of employer and employee. The disease need not to have been foreseen or expected but after its contraction it must appear to have had its origin in a risk connected with the employment and to have flowed from that source as a rational consequence.

"SECTION 25. An employee shall be conclusively deemed to have been exposed to the hazards of an occupational disease when for any length of time however short, he is employed in an occupation or process in which the hazard of the disease exists. The employer liable for the compensation in this Act provided shall be the employer in whose employment the employee was last exposed to the hazard of the occupational disease claimed upon regardless of the length of time of such last exposure, provided, that in cases of silicosis or asbestosis, the only employer liable shall be the last employer in whose employment the employee was last exposed during a period of sixty (60) days or more after the effective date of this Act, to the hazard of such occupational disease, and, in such case, an exposure during a period of less than sixty (60) days, after the effective date of this Act, shall not be deemed a last exposure.

"The insurance carrier liable shall be the carrier whose policy was in effect covering the employer liable on the last day of the exposure rendering such employer liable in accordance with the provisions of this Act."

What do the foregoing provisions mean to members of the medical profession generally or to the legal profession, or to the arbitrators or commissioners? How would the average employer know what occupational disease hazards there are in his employment situation, or what disability or death there may be, now or later, from occupational diseases, or what preventive measure he should use? How would any employee in case of sickness or disability, know whether such sickness or disability is due to occupational disease in his employment, or in case of death, from any sickness or condition during employment, how could his dependents know whether the death was compensable? What background of study of the industries

of Illinois and of their occupational diseases or hazards, or what background of general knowledge or precedent, is there for the application and administration of this new law right now? If to this new law there is given the same liberality of construction, and if the same doctrine as to either direct causation or pre-disposition, or of aggravation, as exists in connection with the Workmen's Compensation Act, then what is the coverage of the new law?

INSOFAR as an occupational diseases law provides compensation for disability or death actually due to occupational disease, definitely arising out of and in the course of the employment, it is in accord with the generally accepted principle of compensation relating to accidental injury, and is generally accepted as right in purpose and principle, and is desirable legislation.

But insofar as such a law, or its application and administration, may impose upon industry payment for sickness or disability, or conditions of old age or disease or death to which all are subject irrespective of employment,—that is, insofar as it may be in effect, health, old age, unemployment or life insurance, merely because of a relationship of employment,—it holds grave dangers to employers, to employment, to employees and the general public:

In that it may be destructive of industry, or drive industry from the state;

In that employment may be materially reduced; and, more particularly,

In that there are great numbers of employees who have infirmities or diseased conditions, etc., not due to employment, who probably would be wage earners during a normal period of life, but if an occupational diseases law makes probable the imposition of payment therefor by industry, would find their employment rendered doubtful or impossible.

The principle of such a law is, therefore, sound and commendable. But the proper application and administration of such a law will require the sincere and intelligent cooperation of all concerned with its operation and its administration. The provisions of a particular law, but more particularly, the general knowledge of the entire subject of occupational diseases, and even more particularly, an intelligent and proper application and administration of such a law, are of even greater importance and concern.

It may be said that such a law merely extends the provisions of compensation acts relating to accidental injury and should, therefore, not be difficult of application or administration. The fact is that there were comparatively few practical difficulties with compensation acts relating to accidental injury, in that there was an extensive background of general knowledge and understanding, and an accidental injury is essentially different from an occupational disease. An accident is a definite occurrence, at a definite time and place which ordinarily can be definitely ascertained and established by witnesses to the occurrence and by immediate symptoms and objective conditions, and the results of an accidental injury are ordinarily of reasonably immediate occurrence and subject to reasonably certain determination as to extent and disability. So, also, the matter of proper medical treatment for accidental injuries and the determination of results or disability is within the knowledge and experience of the medical profession generally, and, as well of those having to do with application and administration of compensation acts.

On the other hand, the matter of sickness generally, and perhaps more particularly the matter of an occupational disease, is entirely different from an accidental injury in that it may come on insidiously and obscurely, without knowledge as to its inception and occurrence, and its duration or development may be over a period of many years before any disability results, and the differentiation between an occupational disease arising out of employment from a disease or condition to which anyone may be subject irrespective of employment, and which may be of essentially the same character or have similar symptoms, clinical aspects and results, is a differentiation requiring the most expert medical diagnosis.

It may be conservatively stated that there is comparatively little knowledge by employers or employees, or the medical or legal professions, or Industrial Commissions, regarding occupational diseases generally, and that no one knows all of the occupational diseases that may be incident to industry, or their diagnosis, or the determination of disability resulting therefrom.

Many illustrations of the difficulties of differential diagnosis and the determination of disability, might be given. One such would be illustrative of the knowledge of silicosis, a greatly over-publicized but by no means the most important occupational disease. It is a matter of rather general information that within the last two or three years —during the so-called "epidemic" of law suits for alleged occupational disease, principally only for silicosis—there were many cases in which employees received large judgments and large voluntary settlements, upon reports of medical examinations, and many who were refused employment upon medical examination, who are now being accepted as good medical risks.

Dr. J. A. Britton, Supervisor of Medical Service of the International Harvester Company, Chicago, who has made an extensive study of occupational diseases for a number of years and is recognized as a national authority, in conducting the "Open Forum" at the Detroit Convention of the American Foundrymen's Association, May 8, 1936, said:

"But what about occupational disease? There is much to do about occupational disease, yet if I were called upon tomorrow in Chicago to get expert knowledge by some physician who knows by actual experience what to do in occupational disease cases, I could probably find a few men who would know about one or two types of occupational disease, no one who had anything like a comprehensive knowledge of occupational diseases, and no one at all who could be of much help with some of the new hazards that are associated with new manufacturing processes."

THE matter of background, knowledge and precedent, is always of interest in considering any new legislation and its problems, and for that reason, and also to show the necessity and value of adequate study and investigation prior to legislation—and which was not made prior to the new Illinois law but will yet have to be made—we shall briefly consider the prior legislation on oc-

cupational diseases within the State of Illinois.

While there has been legislation on occupational diseases in Illinois since 1911, and certain studies and investigations were made as a basis for that legislation, and certain occupational diseases have been compensable since 1923 which, among others, come under the new law, it might seem that in all respects this new law might have a sufficient background of precedent and general knowledge so that it should not present serious problems as to application and administration. Yet the fact is that it does, in effect, open up an entirely new field in which there is no general knowledge by employers, employees, the medical or legal professions, or by the arbitrators and commissioners who will be called upon to administer it. And the occupational diseases and hazards in industry and the necessary preventive measures are generally unknown, even to the limited few who assumed to represent the employers and the employees of the State and drafted and submitted an agreed bill, or to the Legislature who enacted that bill into law without discussion, change or amendment.

Prior to 1911, and indeed until the present time, there was not a common law right of action for occupational diseases, and there was no common law background, precedent or knowledge.

The legislature by joint resolution, authorized the appointment by the Governor of an Occupational Diseases Commission, and the Governor appointed an extremely able Commission, and among those working with it, were Dr. Alice Hamilton, Dr. E. R. Hayhurst, Dr. George L. Apfelbach, Dr. Walter S. Haines, Dr. W. H. Allport, Dr. O. F. Funkhouser, Dr. T. E. Flinn, Dr. P. F. Becker, Dr. Matthew Karasek, Dr. Peter Bassoe, Dr. George E. Shambaugh, Dr. Francis Lane, and Dr. John B. Ellis.

That Commission made a preliminary report in 1909, and by legislative action was extended for two years, with an appropriation for further study and investigation. It submitted an extensive report in 1911 and the legislature enacted the bill submitted, which became the Occupational Diseases Law of 1911. The Commission made a very extensive study and report of occupational diseases in what were then considered to be hazardous employments or industries.

In its report, the Commission strongly urged that it be continued for another two years with an appropriation of $30,000 for study of other industries and other occupational diseases, and for providing information to employers and employees, and to the medical profession, as to any such diseases, and their prevention and treatment. That was not done, and no similar study or investigation has been made since that time, and none was made as a basis of the new Illinois law.

The law of 1911, as reported by the aforementioned Commission and enacted by the legislature, contained two principal sections, 1 and 2. Section 2 referred to certain specific employments, which had been thoroughly investigated and reported upon by the Commission: those in which lead or its derivatives were used; those in which lead, brass or zinc were used or smelted; and those in which poisonous chemicals, mineral or other substances were manufactured or used in harmful quantities or under harmful conditions. Other sections of the act made specific provisions for the prevention, etc., of occupational diseases in such employments. Occupational diseases in such industries, as lead poisoning, etc., were definitely known diseases of which definite diagnosis could be made and definite preventive measures could be used; and it is most important to know that, as a result of the studies and reports of that Commission and of the general knowledge relating to such diseases and methods of prevention, such diseases have all but been eliminated. An accurate knowledge as to the existence of actual occupational diseases and of means of prevention is of fundamental importance, and is most essential to the solution of occupational disease problems.

The occupational diseases arising under Section 2 of the Compensation Act were made subject to compensation in 1923, and, largely because of the study that had been made by the Occupational Diseases Commission and the preventive measures provided, and the general knowledge made possible by the Commission, no serious problems as to such diseases were presented and, as previously stated, they have been almost eliminated.

As to other employments or industries, for a study of which the Occupational Diseases Commission in 1911, urged an extension of its work for two years, with an appropriation of $30,000 the Commission could only make the general and vague provisions in Section 1 of the 1911 Act. It is interesting and informative to refer, very briefly, to its provisions and what resulted from them.

Section 1 of the 1911 Act (all of which Act was repealed by the new law) provided:

"That every employer of labor in this State engaged in carrying on any work or process which may produce any illness or disease peculiar to the work or process carried on, or which subjects the employees to the danger of illness or disease incident to such work or process, to which employees are not ordinarily exposed in other lines of employment, shall, for the protection of all employees engaged in such work or process, adopt and provide reasonable and approved devices, means or methods for the prevention of such work or process."

Section 15, which in 1923 became Section 15(a), provided a right of action for damages for disability or death due to a wilful violation of or wilful failure to comply with Section 1.

Well, no one knew what that meant—neither employers, employees, the Factory Inspection Department, nor the Commission that drafted it. There were no suits under it for some 15 years, and then only a few, until in two cases in 1933 the Supreme Court sustained judgments for damages in silicosis cases. And then, within approximately a year, there was a veritable epidemic of law suits and claims aggregating many millions of dollars, spreading to all industries and to all sections of the State, and greatly endangering industry and employment—and that situation developed because of the very vagueness and uncertainty of the provisions of said Section 1. Some lay testimony as to dusts and some medical evidence as to causation, predisposition or aggravation could always be secured, and verdicts of approximately $10,000 to $20,000 resulted. That Section was held unconstitutional because it was so vague and uncertain that it was in violation of due process of law, and there was no background of common knowledge or precedent such

as would make its provisions understandable or applicable. The thousands of claims and suits that developed in little more than a year were practically all based upon silicosis or pneumoconiosis from the alleged inhaling of silica and other dusts. But those diseases have been greatly over-publicized, and will be found to be relatively simple, and not extensive or numerous, as compared with those which will later be found and for which claims can well be made under the new law within the next few years.

The discussion thus far may at first seem unduly long, but a careful consideration of it will show its fundamental importance. It is a necessary basis for the suggestions I shall make, and for an understanding of the importance of things which all concerned with the new law should immediately proceed to do.

A VERY large part of the problems under the new law are of a medical nature. The determination of what are occupational diseases, their diagnosis and differentiation from diseases and conditions not due to employment, the determination of disability, etc., are primarily medical problems. Unquestionably there will be a tendency to attribute any diseased condition to employment, and the application and administration of the new law will depend very largely upon the extent of knowledge of the medical profession as to the diagnosis and differentiation of diseases due to employment. The medical profession at this time is not adequately prepared to meet this great obligation. The medical profession, through associations and committees, must make an intensive study of occupational diseases, and medical information must be made generally available. This situation is a direct challenge to the medical profession, and it must be met if the new law is to be properly applied and administered in the interests of employers, employees and the public.

There must be an intensive and intelligent study of the industries of Illinois to determine what occupational diseases and hazards there are in employment; and then preventive measures must be used. The prevention of occupational diseases is of greater importance than a palliative procedure of payment for disability or death after disease has occurred. Employers should realize that the cost of proper study of industry and its processes, and then of prevention, is more important and will be infinitesimally less costly than a procedure of just paying for cases that may develop. The best efforts of those skilled in engineering and in prevention, and of those engaged in preventive medicine, and of those who have specialized in occupational diseases and occupational disease hazards, must be enlisted in this great work.

There must be medical inspection, direction and examination, to the end that those having diseased conditions, or being susceptible to particular diseases or hazards, shall not be placed in dangerous work; and so that those beginning to be diseased may be placed in work removed from such hazard, in order that the existence of disease or hazard may be prevented.

There must be cooperation by those representing employers and those representing employees, because they have a great common interest and concern under this new law and in the determination of diseases and hazards and their prevention.

THE Industrial Commission, under the Health, Safety and Comfort Act, effective May 16, 1936, has the duty and power to provide reasonable protection to the lives, health and safety of all those employed, and to make rules for prevention of occupational diseases, and to appoint advisory committee or committees with the ablest possible membership, including members of the medical profession: of those who have the greatest experience and knowledge of occupational diseases and occupational disease hazards; of those who have been concerned with preventive measures and preventive medicine, and of those of engineering ability. Such committee or committees should have adequate funds thoroughly to study all industries in Illinois and determine all occupational diseases, and all occupational disease hazards and their elimination and prevention, and all such information should be made available to employers, employees, the medical profession, and all concerned with the new law.

Employers, in their respective industries, and through their associations, should also make such a study under proper direction, and should cooperate in every possible way with the Industrial Commission and its advisory committee or committees.

If my discussion shall contribute something to the understanding of the general problem; if it shall help to cause study and action which will tend to prevent and eliminate occupational diseases and hazards, and shall in any way promote proper cooperation and proper application and administration of the new law, I shall feel that I have been of some little service.

Textbook of General Surgery

Book Review by

JAMES T. CASE, M.D., F.A.C.S.
Chicago

THIS volume*, dedicated to Dr. Evarts A. Graham, is the direct outgrowth of a formal recitation and lecture course in surgery given by the authors at the Washington University School of Medicine. The subject matter is presented from a physiological point of view, with the hope that the clinical features, including particularly the principles underlying treatment, will be better understood. Details of non-operative therapy have been fully described. Operations, although discussed in principle, have not been described in detail except in a few of the most common instances. It seems wise that these details should have been omitted in a textbook of this type, as it would render the text very cumbersome. The authors have included excellently written chapters on gynecology and genito-urinary surgery. Although the material is necessarily brief in order to be included in a single volume textbook, it seems very well planned. The illustrations are abundant and really illuminate the text. There is a gratifying crispness to the text, which is strictly up-to-date. One searches in vain for a slip-up on the part of the authors on some new feature of surgical diagnosis or therapy.

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* TEXTBOOK OF GENERAL SURGERY. By WARREN H. COLE, M.D., F.A.C.S., Professor of Surgery, University of Illinois College of Medicine, and Robert Elman, M.D., Associate Professor of Surgery, Washington University School of Medicine, Saint Louis. Pp. 1031; illustrations 559; price $10.00. D. Appleton-Century Company, Inc., New York, 1936.

The History of Industrial Medicine and Occupational Diseases

—An Historical Outline of Occupational Diseases, Industrial Hygiene and Surgery, Traced from Early Antiquity to the Great Depression of the Twentieth Century—

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Industrial Hospitals

THE word "hospital" has its derivation in the Latin adjective *hospitalis* and the noun *hospes*, meaning a host or guest. The three words, hospital, hotel and hospice are derived from the same source, but have been given different connotation in our language.

Hospitals, called Halls of Healing, were founded in China in 651 B.C. Herodotus says there is evidence that hospitals flourished for the sick in ancient Greece. In 226 B.C., King Asoka erected a hospital in India. Alexander the Great established medical houses for the sick and wounded in 300 B.C. During the early Christian era (31 A.D.) shelters were erected for the sick and crippled at the "Pool of Bethesda."

The first definite military hospitals were established in Rome during the reign of Hadrian (117-138 A.D.) St. Basil built hotels in Caesarea, and asylums for the sick and destitute were established by Justinian, 369 A.D., and Fabiola in Rome, 370-80 A.D.

The Hotel Dieu was founded in the seventh century (651 A.D.) at Paris by the Bishop of St. Landy. In England, St. Gregory's Hospital was founded in 1084 A.D. by the Archbishop La Franc of Canterbury; St. Bartholomew's at Smithfield in 1137 by Prior Rahere; St. Thomas' Hospital in 1215, by Peter, Bishop of Winchester.

Pope Innocent III summoned Guy de Montpelier to found for him during the thirteenth century the Hospital of the Holy Spirit at Rome. This hospital was the mother hospital for the world. The Hospital St. Louis was founded in 1607 in Paris by Henry IV; it was named after Louis IX, a crusader who died of plague.

Johnde Dios, a Portugese, of the Order of the Brother Hospitallers, established hospitals with other pious men, the outcome being the founding of the "Order of Charity."

The first hospital in the Western Hemisphere was erected on the site where Montezuma met the conqueror in Mexico City, in the sixteenth century; Cortes built the structure and named it the Hospital of Jesus (1524). Canada's first hospital was built in 1639.

In America the following early hospitals were established: the Pennsylvania Hospital in 1751, which Benjamin Franklin actively supported; the New York Hospital in 1771, conceived by Trinity Church; and the Massachusett's General Hospital, in Boston in 1811, which became famous in 1846 when Morton introduced ether anaesthesia. The first emergency hospital service was established in New York in 1875, and was known as the Chamber Street Hospital. National Hospital Day is celebrated on May 12, the anniversary of Florence Nightingale's birthday.

The guilds in the middle ages rendered assistance to their members in case of illness. The carpenters and masons of Florence had their own hospitals, probably the earliest recorded industrial hospitals.

The modern industrial hospital is a result of necessity. Industries such as mining and lumbering in remote regions needed hospitals for their sick and injured employees. The great railroad systems in America established their own general hospitals, as well as emergency hospitals, at points along their lines where the greatest hazards existed. Then came the emergency hospitals of the steel industries, manufacturing plants of all kinds, construction outfits, department stores, where first aid and the principles of industrial medicine and surgery were skillfully administered. Some of the great railroad hospitals in the United States, maintained by the railroad companies for sick and injured employees, are notable examples of hospital efficiency. One noteworthy example is the Southern Pacific General Hospital of San Francisco, where Chief Surgeon W. B. Coffey and Chief of Medicine P. K. Brown are early workers in industrial medicine.

Industrial Hospitals

AMONG outstanding industrial hospitals are those of the following: Pennsylvania Steel Co., Steelton, Pennsylvania; The Norton Co., Worcester, Massachusetts; Thomas A. Edison Co., Orange, New Jersey; Western Electric Co., Chicago; National Cash Register Co., Dayton, Ohio; American Rolling Mills Co., Middletown, Ohio; Winchester Repeating Arms Co.; McCloud River Lumber Co., McCloud, California; Ford Motor Co., Detroit, Michigan; Colorado Fuel and Iron Co.; Endicott-Johnson Co., Binghampton, New York; Carnegie Steel Co., Clairton, Pennsylvania; Standard Oil Co., Richmond, California; American Smelting & Refining Co.; Johns Manville Co., Lompoc, California; U. S. Steel Corporation; Bethlehem Steel Co., San Francisco, California; United Fruit Cos., Jamaica, West Indies; Fairbanks, Morse & Co., Beloit, Wisconsin; Youngstown, Sheet & Tube Co., Youngstown, Ohio.

Occupational Disease Clinics

THE Milan Clinic, first in the world, was founded in 1910 for the scientific study and teaching of occupational diseases. The director, Professor

Luigi Devoto, has made this clinic internationally famous. It comprises consulting rooms and wards for patients affected with occupational diseases, some 14 laboratories for research, a library, museum and lecture rooms. Many scientific contributions have come from this clinic—notably researches on over-exertion, dust diseases, pellagra, lead, etc. The clinic offers a graduate course of instruction of one year leading to a diploma for physicians studying clinical and scientific industrial diseases. It publishes not only *La Medicina del Lavora,* founded by Dr. Devoto in 1901, but also popular lectures and bulletins on industrial hygiene for factories and workers. The first industrial clinic in Great Britain was established by Cadbury Brothers in 1905 at Brownsville.

In 1919 an Industrial Clinic was founded in Naples, Italy. The same year an industrial hospital and dispensary was established at Shanghai, China, by Dr. H. W. Decker, for cotton workers.

The pioneer clinic in this country was established by the Cornell Medical School Outpatient Department in 1910 by Dr. W. Gilman Thompson to study and treat occupational diseases. It was discontinued in 1916.

The Sprague Memorial Institute of the University of Chicago was founded in 1911, with Dr. E. R. Hayhurst as its first Director. It has made important contributions concerning compressed air disease, and brass and lead poisonings. The University of Pennsylvania organized an occupational disease clinic at its hospital in 1914, and has contributed data on dust investigations and on pulmonary diseases of potters and cement workers.

Clinic for Industrial Diseases, Milan, inaugurated March 10, 1910; Director Professor Luigi Devoto

The first effort to study industrial diseases in the Massachusetts General Hospital, Boston, Massachusetts, was made in 1913 by a pioneer social worker, Miss Susan Holt, eventually resulting in the establishment of an industrial clinic in 1916, with Dr. Wade Wright as the Chief.

The New York City Health Department established a clinic in 1915 devoted to occupational diseases, under the leadership of Dr. S. S. Goldwater, Commissioner of Health. Its contributions include many publications for factory physicians.

At Columbus, Ohio, the State University College of Medicine in 1915 developed a clinic under Dr. Emery R. Hayhurst for the study of occupational diseases. Dr. W. G. Thompson in 1918 organized in New York City a clinic for the functional re-education of industrial and war cripples.

The Joint Board of Sanitary Control of the Cloak and Suit Industry of New York City established in 1920 a clinic for workers in that industry, which is supported by joint contributions from employees and employers. Dr. George M. Price was the first Director.

In 1934 New York University and Bellevue Hospital opened a special clinic and laboratory devoted to occupational diseases.

Professor Luigi Devoto

Research in Occupational Hygiene Abroad

AUSTRALIA: Central Office of Preventive Medicine (founded 1921), Division of Industrial Hygiene, Dr. Robertson, Director.

FRANCE: Institute of Occupational Diseases of Lyons University. Societe d'hygiene publique industrelle et sociale (founded 1925). Institute of Industrial Hygiene and Medicine, 1933, Paris School of Medicine, France.

JAPAN: Institute for Industrial Research at Kurasiki (founded 1921), Dr. Gito Teruoka, Director.

RUSSIA: The Obuch Institute of Professional Diseases was founded at Moscow in 1923. There are Institutes of Occupational Diseases at Leningrad and Karkov.

SOUTH AFRICA: South African Institute of Medical Research and Miners Phthisis Bureau.

SWITZERLAND: Institute of Legal Medicine at University of Zurich, H. Zangger, Director. Health Organization of League of Nations, Geneva, 1923.

Other research institutes in industrial hygiene were developed particularly in: 1893, in Amsterdam; 1894, in Milan; 1900, in Munich; 1903, in Berlin; 1908, in Budapest; 1909, in Vienna; and 1924, the Institute of Social Medicine, in Zagreb, Jugoslavia.

GREAT BRITAIN: Industrial Fatigue Research Board, London, 1918; National Institute of Industrial Psychology, London, 1921; and H. M. Factory Inspectors' Laboratories, London.

GERMANY: Hygienic Institute, University of Munich, 1879; Institute for Investigation of Industrial Diseases and Accidents, Dortmund, Prusria, 1920; and German Society of Industrial Hygiene, Frankfurt am Mann, 1922.

UNITED STATES: Bureau of Mines at Pittsburgh, Pennsylvania, 1915; Carnegie Foundation; Henry Phipps Institute, of Philadelphia, Pennsylvania; Industrial Health Service Bureau, Chicago, 1921; Life Extension Institute; and National Safety Council, Chicago.

Investigators Throughout the World

AUSTRALIA: Pneumonoconiosis and lead poisoning: D. G. Robertson, of Melbourne; Badham, of New South Wales; H. W. Armit, of Sydney; Smith and W. Summons, of the Bendigo Mines.

AUSTRIA: Occupational hygiene: Rambousek; E. Brezini; H. von Schroetter; Sternberg; H. S. Jellinek; L. Bohler, of Vienna.

BRAZIL: In the field of forensic medicine and industrial diseases and accidents: A. Puxato; F. Favero; and L. Ribeiro, of Rio Janeiro and Sao Paola.

BELGIUM: Industrial and compensation: Prof. Malvoz, at Liege; De Hermans, of Mons; Deladriera, of Brussels; De Net, at Chourg lez Mons; Mund, of Louvain; and L. Dejardins, of Brussels; M. Stassen, of Liege; M. De Laet; D. Gilbert, of Brussels.

CZECHOSLOVAKIA: Industrial physiology and social medicine: J. Lowy, of Prague.

DENMARK: S. K. Gudjonsson, of Copenhagen.

ENGLAND: Mine and factory hygiene and fatigue: J. S. Haldane; L. Hill; T. Oliver; T. M. Legge; E. L. Collis; Sir Arthur Whitelegge, of London, First Factory Inspector; G. P. Crowden, of London, and E. Atzler of Dortmund; P. White, of Manchester; F. Shufflebotham; C. S. Myers; Dr. J. C. Bridge; S. E. L. Middleton, of London.

Industrial Dentistry and Nursing

IN ANTIQUITY, it is said that Cascellus, 80 A. D., was the first to practice dentistry. The Etruscans were wonderfully skilled in dentistry, especially in the making of false teeth and bridgework. Guy de Chauliac, 1300-70, the eminent surgeon, in his great work summarizes the dentistry of his time. One of his pupils, Pietro d'Argelata, was skilled in dentistry.

Giovanni d'Arcola, of Padua, known as Arculanus, was the first to use gold leaf for filling teeth in 1450.

The earliest book on dentistry was the ARTZNEY BUCHLEIN, published in Leipzig in 1530. A. Pare, the French barber surgeon, was influential in placing dentistry on a physiological basis in 1561.

Bartolomeus Eustachius, an Italian anatomist who described the anatomy of the tooth in 1563, wrote an early book on dentistry. The first English work on dentistry was published by Allen in 1685.

Pierre Fanchard, of Brittany, known as the Father of Dentistry, advanced the idea of dentistry as a real profession.

John Hunter, the famous English anatomist and surgeon, in 1771 published his classic work, the NATURAL HISTORY OF THE TEETH, and laid the foundation for the study of comparative dental anatomy.

Systematic dental instruction was first instituted at Guys Hospital, London, in 1799 by Robert Blake. R. C. Skinner, a New York surgeon-dentist, wrote the first American book on dentistry in 1801.

The first dental college was organized in 1840 at Baltimore, Maryland, by Drs. H. H. Hayden and C. A. Harris.

Joseph Fox published his book, NATURAL HISTORY OF THE HUMAN TEETH, in 1803.

The American Society of Dental Surgeons was founded in 1840. Harvard University was the first university to develop a dental department, awarding a degree of D.M.D., in 1868.

America's dental contribution to human welfare was the introduction, by Horace Wells, of Hartford, Connecticut, of nitrous oxide gas as an anaesthetic in 1844, and the demonstration by Dr. W. T. G. Morton, of Boston, of ether anaesthesia at the Massachusetts General Hospital in 1846.

In 1867 Dr. Bruck, a Boston dentist, used a hot platinum wire to illuminate a cystoscope he invented.

John M. Riggs, of New York (1811-1885), first dentist to remove a tooth under nitrous oxide administered by Dr. Horace Wells, described pyorrhea alveolaris (Rigg's Disease) in 1876.

The American Dental Association was organized in 1859, with Dr. W. W. Allport as its first president. The National Board of Dental Examiners began functioning in 1933. The Dental Corps of the U. S. Army was established under Captain John S. Marshall, of Berkeley, California. In 1932 there were 31 Class A dental colleges in the United States.

The industrial dentist is rapidly finding his own importance in the modern industrial health program in many of the larger plants, railroad hospitals and department stores. His services have enhanced the standard of efficiency by eliminating dental foci of infections, providing dental examinations, hygiene, extractions and restorations. Some are full time dental surgeons, while the smaller institutions have the services of part time dentists. In special hazards, where phosphorous, radium and other poisonous elements are employed, dental supervision of the workers' teeth is of great importance. In fractures of the mandible, the professional skill of the dentist in managing such cases places him in first rank as a specialist.

Some American industrial establishments that maintain dental services are: Heinz Co., of Pittsburgh; The J. G. Brill Co.; American Cast Iron Pipe Co.; Mazda Lamp Co.; The Crane Co. of Chicago; The Ford Motor Company, Detroit; Joseph & Feiss Co., Cleveland; J. B. Stetson Co., Philadelphia; John Wanamaker Store, New York; Montgomery Ward & Co., Chicago; National Cash Register Co.; The Norton Co., Worcester, Massachusetts; B. F. Goodrich Co.; Le Blond Machine Tool Co., Cincinnati, Ohio; American Bush Magneto Co.; U. S. Playing Card Co.; Amalgamated Clothing Workers of America, Cincinnati, Ohio; Hood Rubber Co., Watertown, Massachusetts; Endicott-Johnson Co.; Joint Board of Sanitary Control, New York.

A few industrial dentists who have contributed to the welfare of American toilers are: J. B. Beebe, of B. F. Goodrich Co.; Ralph W. Elliott; C. B. Bray; H. M. Brewer, of National Cash Register Co.; A. D. Rood, of American Bush Magneto Co.; Sanford De Hart.

History of Industrial Nursing

DURING the Middle Ages the attendants on the sick in hospitals were the sisters in the various religious orders, such as the Sisters ot Charity and Vincent de Paul.

History gives credit for the origination of trained nursing to the Reverend Theodore Fleidner, of Germany, who in 1833 conceived the idea of training persons for that work; but the world honors Florence Nightingale (1823-1910) as the Englishwoman whose nursing work during the Crimean War in 1854 led to the establishment of the first hospital training school at St. Thomas' in London in 1860. In that same year she published her "Notes on Nursing".

The efforts of Clara Barton to develop nursing for the Union army during the Civil War, and her later influence on the American Red Cross, have placed her among the famous women of America.

The Bellevue Training School, organized by Louise Schuyler in New York, first graduated nurses in 1872. Lillian D. Wald pioneered an important venture when she founded the Henry Street Settlement in New York in 1893. From this practical dealing with social, economic, and industrial conditions developed the visiting nurse.

It was because of the foresight of Isabel H. Robb, of the Johns Hopkins Hospital, that the first National Society of Nurses was organized at Chicago in 1893. The trained nurse probably entered industry in 1895 when Ada M. Stewart was engaged by the Vermont Marble Company to visit homes and care for the sick workers and their families.

The American Nurses' Association was founded in 1896. The industrial nurse, it is said, was pioneered in this country by Anna B. Duncan, of the John Wanamaker Company, in New York in 1897. That same year the Cadbury Company, a chocolate factory in England, established a health department and employed nurses for the workers.

On the Pacific coast in 1900, Dr. R. T. Legge introduced graduate nurses into service in the lumber industry at McCloud, California. State registration of trained nurses began in America in 1903.

The Metropolitan Life Insurance Company, of New York, in 1909 introduced home nursing for industrial policyholders. Adelaide Nutting was the first Professor of Nursing in any university. She established the graduate courses at Teachers' College, Columbia University, in 1910.

The National Organization for Public Health Nursing was organized in 1912. Eight years later it developed a section in industrial nursing, and in 1930 became the industrial nursing section of the National Safety Council. The Chicago Industrial Nurses' club was organized in 1917 with an enrolled membership of 65 industrial nurses.

Annie W. Goodrich, of Yale University, developed the undergraduate education of nurses within a university on a university level, and became first Dean of the United States Army School of Nursing during the Great War. Jane Delano, of Washington, D. C., became outstanding as a national leader in nursing through her work with the nursing section of the American Red Cross and her organization of nurses in the Great War. During the War, Mary E. Lent organized the nursing service of the United States Public Health Service.

The "Nurse in Industry" chapter of Mock's INDUSTRIAL MEDICINE AND SURGERY was written by Mae Middleton, an industrial nurse of Chicago, in 1919.

A Nurses' Registration law was passed in England in 1919. In the same year, from the pen of Florence S. Wright, an industrial nurse, came an early book on INDUSTRIAL NURSING. It was followed in 1933 by V. H. Hodgson's work, PUBLIC HEALTH NURSING IN INDUSTRY.

Among the women who have distinguished themselves as leaders and contributors to the various fields of nursing in the United States are Lavinia Dock; Mary S. Gardner; Diana Kimber; Linda Richards; Elizabeth G. Fox; Edith S. Bryan; and Anna Jammie. Major Julia C. Stinson of the United States Army became the first ranking officer and superintendent of the Army Nursing Corps.

Louise M. Tattershall, R.N.; Carey P. McCord, M.D.; and R. T. Legge, M.D.; have contributed articles on Public Health Nursing in Industry.

Urology

Book Review by

KNOWLTON E. BARBER, M.D., A.U.A.,
Chicago

FOR a practical book on urology this book* is well fortified with numerous photographs and charts—x-ray films, microscopic sections—and, above all, numerous photos of well-demonstrated clinical cases, with pathological specimens to acquaint the physician or surgeon with a vision of the underlying diseased organ or anatomical structure, also the types and technique of the operations best suited to remove or correct the affected parts.

The continuity of the discussion of different urological conditions, their causes, symptoms, diagnosis, pathogenesis, prognosis and treatment, are well organized and of simple working so that one may pick up the book and cover any subject involving the field of urology without reading pages or paragraphs of unnecessary words.

There is valuable information in endocrinology and irradiation which brings this book up to date. The chapters on malignant tumors of the urinary tract and operations on the urinary tract alone make this book one that every practitioner can well afford to have in his library.

This book is well recognized by the Urologist, as it is the standard book used in some of the best medical schools of this country, and we use it at Rush Medical College as our standard book in urology and genito-urinary diseases.

UROLOGY—EDWARD L. KEYES, PH.D., F.A.C.S., F.R.C.S. (Hon. England), Professor of Urology, Cornell University Medical College; Attending Urologist, New York Hospital; Consulting Urologist, Memorial, St. Vincent's and Bellevue Hospitals, New York City; President of the International Urological Society, and RUSSELL S. FERGUSON, A.B., M.D., Assistant Professor of Urology, Cornell University Medical College; Associate Attending Urologist, New York Hospital; Director of the Laboratory of Oncologis Physiology, Memorial Hospital; Assistant Surgeon, Memorial Hospital; Consulting Urologist to the Carpenter Clinic and Homeopathic Hospital, Wilmington, Delaware; and to the New York Infirmary for Women and Children. Sixth Edition. With 343 illustrations in the text, and 17 plates, three of which are in color. D. APPLETON-CENTURY, INC., New York, 1936.

Pathology of the Pneumoconioses

—Silicosis; Silicosis and Tuberculosis; Asbestosis; Siderosis; Anthracosis—

OSCAR AUERBACH, M.D.,
Staten Island, New York

PNEUMOCONIOSIS has been defined by Wilson as a disseminated fibrosis of the lungs which is caused by the habitual inhalation of dust in the course of various occupations. There are three main types of dust that produce singularly characteristic forms of fibrosis. Silica dust produces silicosis; iron dust, siderosis; and asbestos dust, asbestosis. Coal dust *per se* does not produce fibrosis unless it is inhaled along with siliceous material.

Although pneumoconiosis was known to Ursinius,[1] who, in 1652, described two forms of mountain sickness caused by the inhalation of dust, it was not until the past two decades that the greatest progress in the study of this disease was made. This has been accomplished by the study of pathological specimens, animal experimentation, examination of dusts and periodic roentgen examination. There are still a number of questions to be clarified for the better understanding of the disease.

Silicosis

SILICOSIS is by far the most frequently encountered type of pneumoconiosis. It is found chiefly in men who have worked as sandstone masons, sand blasters, as miners of gold, tin, lead, and in other occupations where silica dust was present. Lubbenau,[2] in an attempt to determine which mineral dusts were harmful to the lungs, exposed animals to 150 grams of each of the 28 different types of dust. He found marked lung changes as a result of the inhalation of brass, dolomite and lead sulphide; slight changes from porcelain, cement, sandstone, road dust and glass; and minor changes from marble, brick and granite. He could detect no evidence of pulmonary damage after inhalation of coal dust. These experiments, however, according to Ickert[3] and others, had one important fallacy: the conditions under which the changes were produced in animals were entirely different from those in the human being. Moreover, the human being does not inhale as large amounts of dust as were used in the experiments. Ikert has estimated that in a 40-year period, underground copper slate miners inhale with the air only from 48 to 480 grams of dust.

The production of silicosis in the lungs depends upon the following factors, some of which are still the subject of controversy:

1. Chemistry of the dust.
2. Mechanical irritation or physiochemical reaction of the dust.
3. Length of exposure.
4. Concentration of dust particles in the air.

1. CHEMISTRY OF THE DUST. There are three types of siliceous rock encountered in industry. The first kind is composed almost entirely of some form of silicon dioxide (sand, sandstone and flint). The second contains silicon dioxide and one or more silicates (granite cutting). The third type is composed of silicates (kaolin, feldspar, mica and talc). Jephcott[4] believes that the dusts of the first kind are most dangerous, and those of the third type least dangerous. Until the recent studies of Jones[5] it was generally accepted that only uncombined (free) silica was capable of producing silicosis. Cummins[6] justifies this belief by the recovery of a high percentage of silica from the ash of silicotic lungs and the finding of bright particles in the same lungs, which were interpreted as free silica.

Jones, analyzing and comparing the rock of the mines of Johannesburg, in which locality silicosis is common, with those of Kolar, where it is uncommon, found that in Johannesburg the rock is rich in sericite, whereas the rock of Kolar is poor in this substance. The bulk of the mineral residues obtained from 29 silicotic lungs was composed of sericite, a silicate of aluminum and magnesium, with free silica present in a lesser amount. Gardner[7] does not completely accept the views of Jones, although he admits that his arguments are convincing and that the presence of sericite in certain localities coincides with the occurrence of silicosis. He points out the fact that there are industries handling pure quartz in which individuals, exposed to this dust, develop silicosis. Gardner[8], Kettle[9] and Mavrogordato[10] have produced silicosis in animals similar to that in humans after exposure to pure quartz.

Cummins,[6] feels that the criticism of Jones does not invalidate the findings of Gardner, Kettle and Mavrogordato simply because the dust used in their experiments contained mixed constituents. Still more recent studies tend further to disprove the contention of Jones. Kettle[11] has shown that the introduction of kaolin (aluminum silicate) into the lungs of guinea pigs did not produce pulmonary fibrosis in 500 days. Jephcott,[4] who examined the lungs of individuals who had died of silicosis, was unable to find any sericite; on the other hand, he recovered silica compounds in the lung ashes corresponding to the dusts inhaled during life. Even before the work of Jones, Watkins-Pitchford and Moir[12] found that the silicotic lungs of South African miners contained not only free silica but also sericite.

Moir[12] in a statement from the Miners Phthisis Medical Bureau and Department of Mines, reports the presence of silicosis in miners in which no free silica could be found, and regards this as positive evidence against the theory that free silica is the important element in producing silicosis.

2. MECHANICAL IRRITATION OR PHYSIOCHEMICAL REACTION OF THE DUST. Whether pulmonary fibrosis results from the chemical action or the physical irritation of the siliceous material is still a debatable question. It has been rather widely held that it is the mechanical irritation resulting from the inhaled particles that is the important factor in the production of silicosis.

Mavrogordato,[13,14] comparing the effects of inhalation of coal, shale, "flue" dust, precipitated silica, quartz and flint dusts, found that the most

marked lung changes developed from the harder dusts. Landis[15] found that the severity of the lung lesions varied in accordance with the hardness, sharpness, and chemical composition of the dusts. Oliver[16] has maintained that it is entirely the physical character of the dust which is important, that soft coal is less harmful than hard coal, and that the latter is less harmful than rock; dust from silicia, on the other hand, is more harmful than rock. Watkins-Pitchford[17] also has attributed the injurious quality to the physical property of the dust, i.e., its sharp cutting particles. Watkins-Pitchford and Moir[12] have shown that the greatest number of particles of foreign matter

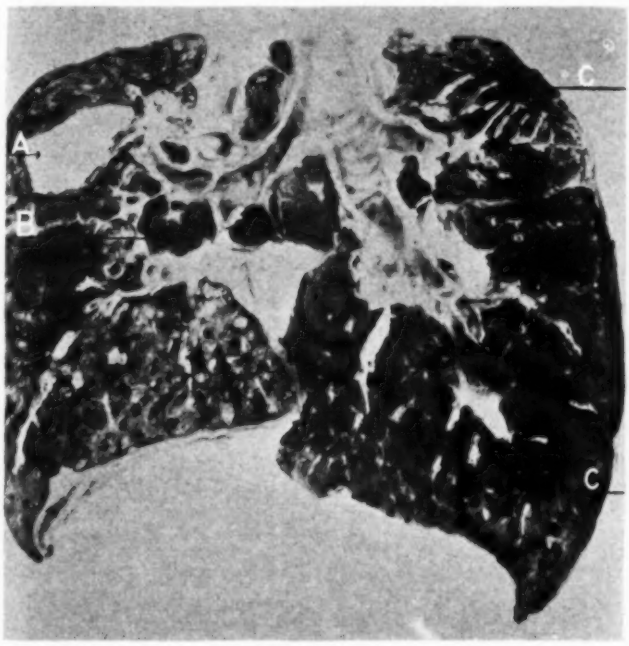

Fig. 1. Silico-tuberculosis—posterior view—(gross specimen). There is a large tuberculous excavation in the left upper lobe (A). Note large silicotic tracheobronchial lymph nodes (B). There is extensive nodular parenchymal involvement (C)

in pigmented lungs are smaller than one micron in length, and that the prevailing form of the particle in such lungs is narrow and elongated, with one or both ends sharp and pointed. Mavrogordato,[14] refuting the arguments that hardness and sharpness of dust cause fibrosis has pointed to the work of McCrae[18] and Watkins-Pitchford[17] and states that when particles are so small (from five microns downward) hardness and sharpness are of no significance; the particles do not penetrate the tissue, but the tissues take up the particles.

Collis,[19] Haldane[20] and Mavrogordato[14] feel that it is the chemical action of the solution of free silica rather than mechanical irritation that produces fibrosis. Gye and Purdy[21] believe that the silica particles in the form of quartz are slowly dissolved in the alkaline fluids of the body and liberate small quantities of colloidal silica, an irritant which causes fibrosis. They have demonstrated that the intravenous injection of colloidal silica in large doses results in rapid death, whereas small doses produce fibrosis. Gye and Kettle[22] have observed that the intravenous injection into animals of small amounts of colloidal silica leads to fibrosis in the liver and kidneys. Cummins[6] feels that the solution theory would be much easier to defend if the views of Jones were correct, because sericite and other fibrous silicates are more soluble than uncombined silica.

3. Length of Exposure. According to reports, the length of exposure to dust before silicosis results varies from a few months to 20 years. Acute silicosis would be that form which develops in a period from a few months to four or five years after exposure to dust.

Gardner,[23] believing that silicosis will develop if a sufficient quantity of fine dust is inhaled over a sufficient period of time, has been able to reproduce in guinea pigs a typical nodular disease in 12 months by eight hours' daily exposure with concentrations of approximately 200 million particles per cubic foot of air. Arnold[24] (1885) exposed guinea pigs, cats and dogs to very extensive amounts of dust over variable periods of time. The changes observed were those of bronchitis and bronchopneumonia followed by a typical nodular formation. In many instances this nodular formation occurred after two years of constant exposure. Ickert,[3] criticizing the work of Arnold, pointed to

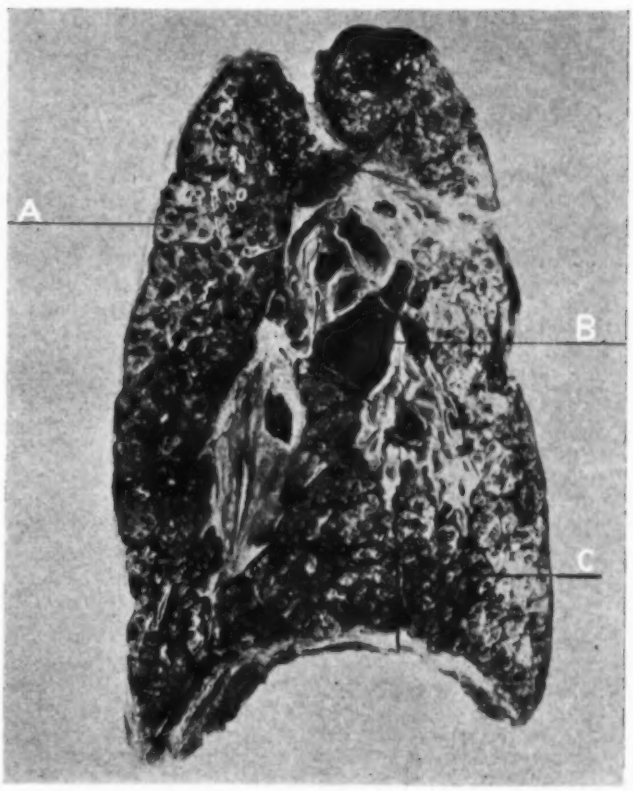

Fig. 2. Silico-tuberculosis—Section through left lower lobe, posteriorly—(gross section). (A)—There are silicotic nodules surrounded by an area of caseous pneumonia. Note the extensively enlarged silicotic tracheo-bronchial lymph nodes (B). (C)—Silicotic nodules in the pulmonary parenchyma

the findings of Willis[25] that the lymphatic system of the guinea pig differs basically from that of the human being, and emphasized the fact that man is not exposed to as large amounts of dust as utilized in experiments. The length of time necessary for silicosis to develop will depend upon the character of the dust, the size of the particles, the number of hours of exposure per day and the concentration of dust particles in the inhaled air.

Watkins-Pitchford found that the average duration of exposure in the South African gold miners

was 9.6 years. Tattersall[26] observed it to be slightly more than 10 years in coal miners. Sutherland and Rivers[27] found the minimal exposure to be six years in the stone crushing industry and Patschkowski[28] and Koelsch[30] first observed changes in porcelain workers after 10 years of exposure, although it was more frequent after 20 years. Chapman[31] reported three cases of silicosis more marked than usual after exposure to an alkaline dust of silica, respiratory symptoms and developing eight, 21 and 29 months respectively after exposure. In two of the cases the diagnosis of silicosis was confirmed by autopsy. These individuals had worked at machines mixing silica and soap and the author believed that the acceleration of the process of silicosis was due to the formation of silica hydrosal in the presence of an alkaline dust. MacDonald[32] also has reported two cases of short duration in young females exposed to silica and soap powder.

The size of the particles inhaled greatly influences the time within which silicosis develops. Lanza[33] is of the opinion that the harmful effects

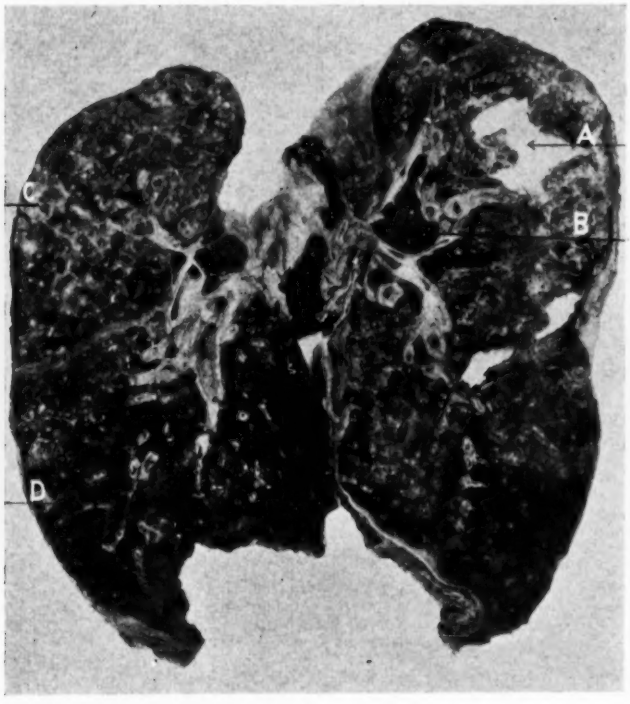

Fig. 3. Silico-tuberculosis — posterior view—(gross specimen). Note the large tuberculous excavation (A). There is an extensive silicotic involvement of the tracheo-bronchial lymph nodes (B). (C)—Demonstrates a silicotic nodule surrounded by an area of caseous pneumonia. Note the silicotic nodules surrounded by an area of caseous pneumonia. Note the silicotic nodules in the substance of the lung (D)

are produced when the particles are in a fine state, that is, less than 10 microns in diameter. (As stated, McCrae[18] has shown that most of the dust taken up by the alveoli is less than five microns in diameter.) Smyth and Iszard,[34] who used dark grounds as well as direct illumination, calculated that from 60 to 80% of countable particles in the lungs of men and animals are from one to five microns in diameter, the most frequent size being one micron. If it is true, as shown by Mavrogordata[13,14] that cells take up dust particles rather than that dust particles penetrate the tissues, then it is understandable that the greater the number of small particles inhaled the more rapidly will the lungs become damaged. Watkins-Pitchford holds that particles gaining access to the lung tissues cannot be much more than 0.5 to five microns in size, because the phagocytic cells lining the alveolar wall cannot engulf larger particles and carry them from the air sacs to the lymphatic vessels. Smyth and Iszard,[34] have also shown there is no evidence of great accumulation of ultramicroscopic particles in the lung and that such particles are much less apt to reach the lung cells than those of somewhat larger size.

The question of the length of exposure is complicated by the fact that pulmonary fibrosis may develop years after men leave their occupation. Britton and Head[35] have reported four cases of men who left their occupation clinically free, and yet many years later developed silicosis. One individual, after a four months' exposure, showed the first signs of silicosis 22 years afterwards. Britton and Head state, therefore, that apparently silicosis may develop after a relatively short period of exposure. They point out that the process may be one of progressive fibrosis, and that symptoms appear late because of the length of time necessary for the disease to develop. Similar observations have been made by Lawson, Jackson and Gardner.[36] In a survey of 17 men who

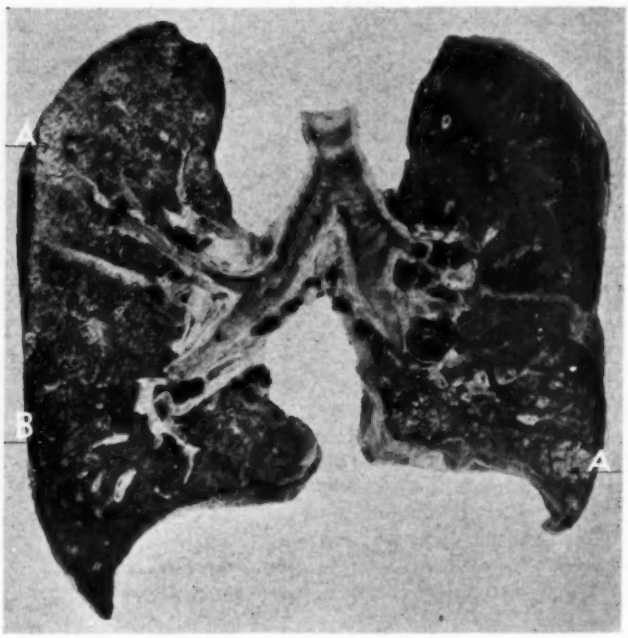

Fig. 4. Silico-tuberculosis—posterior view of lungs —(gross specimen). There are several areas of caseous pneumonia present (A). Note extensive silicosis of lungs (B)

operated air drills in mines laden with iron ore and silica for a period of four to five years, it was found that distressing symptoms of lung fibrosis first appeared after they had discontinued mining and had lived from four to eight years in apparently good physical health. Watkins-Pitchford[37] observed long-service miners who took part in the world war and were non-silicotic when they left their occupation, but who presented the condition after they had returned from combat. He believes that this late onset of silicosis is dependent upon the continued retention in the lung of a sufficient

number of silica particles, and therefore designates this condition "latent silicosis." Serial roentgenological study has shown that once pulmonary fibrosis has been established the process is progressive. Since most men have continued their occupation for many years, it is difficult to establish the length of exposure necessary for an individual to develop silicosis.

4. The Concentration of Dust Particles in the Air. This depends to a large extent upon the type of machine and process used to break up the rock and the precautions taken to moisten the dust immediately after its formation. If the interior of the hole which is being drilled is kept wet the dust turns into mud. Mavrogordato estimated that the dust count of the air in gold mines was 20 milligrams per cubic meter before precautionary measures were instituted in 1912, after which the count fell to one or two milligrams. The Miners Phthisis Commission of 1912 listed machine drilling, as the most dangerous of mining operations in the production of the greatest number of dust particles. Tramming came second, hand drilling third, and timbering fourth. The process of dynamiting produced a large concentration of dust particles which were thrown into the air at a great distance. In all of these operations, of course, the important factor is the formation of particles small enough to damage the lungs.

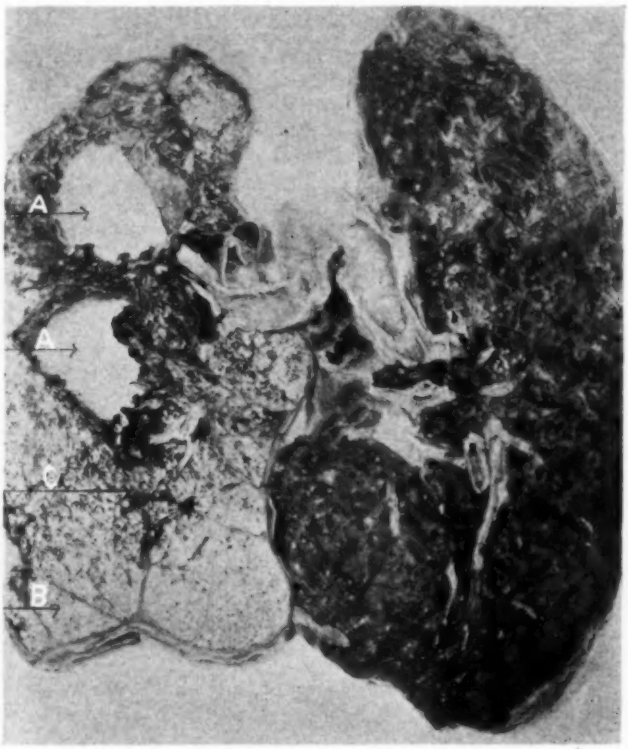

Fig. 5. Silico-tuberculosis—posterior view of lungs (gross specimen). (A)—Extensive tuberculous excavation with rupture through the interlobar fissure (not seen in this view). (B)—Extensive caseous pneumonia surrounding silicotic foci (C)

Examination of workers exposed to varying concentrations of dust has shown that 10 million particles per cubic foot of a dust containing 35% of free silica can be tolerated without danger for a short time. After a longer period of time, however, mild fibrosis results. A low concentration of dust particles containing a high percentage of silica may produce as much harm as a higher concentration of dust with a lower percentage of silica. Lehmann[38] calculated that a dust content of one milligram per cubic meter causes only slight discomfort, five milligrams is tolerable, 20 milligrams is unpleasant, 30 milligrams is irritating, and more than 100 milligrams most disagreeable. Koelsch[30] found that in the porcelain industries where workers developed silicosis the air contained from 22 to 26 milligrams per cubic meter.

Gross Appearance of Silicosis. The lungs of silicotic individuals are observed most frequently in the late stages of the disease. The early changes have been observed in only a few cases. In the very early stages of the disease the interlobular septa are exaggerated, and, scattered throughout the lung tissue in close approximation, are firm islets of gray-black pigment which protrude above the cut surface. The tissue is crepitant and floats in water. As the process continues the islets of gray-black lung tissue increase in size and many fuse, to form larger pigmented areas, the presence of which gives the lung on cross section a gray-black appearance. The tissue is now less crepitant, but still floats in water. Further progression of the process reveals the presence of small, discrete, circular, oval or polygonal light-gray areas of fibrosis lying in pigmented zones. These areas may continue to increase in number and size until they may uniformly stud the cut surface of the lobe. The appearance of these discrete nodules is an indication of the third stage of silicosis. The most striking characteristic of these nodules which may be palpated in the pleura and deeper lying tissues is their firm consistency. Their size varies from one millimeter to two centimeters; occasionally they are larger. The periphery of the fibrotic areas is surrounded by a translucent gray-white capsule. The larger areas extend to the limits of the lobule and may be so numerous that one nodule borders the other, but individual foci are at all times clearly distinguishable. Close scrutiny of any fibrotic area reveals the presence of white rings which represent obliterated blood vessels and bronchioles. In some portions of the lung extensive fibrosis may develop without the presence of small nodules, in which event the cut surface of the lung has an almost homogeneous black color, and there is an inward extension of fibrous tissue from the interlobular septa.

The gross appearance of the lungs of patients who have died with advanced silicosis is striking. When the chest cavity is opened one notes that the lungs are not collapsed, but are voluminous. They weigh between 3,000 and 4,000 grams in contrast to the weight of normal lungs, which is between 900 and 1,000 grams. The pleurae are thickened and the pleural space is often obliterated. The thickened septa are seen upon the pleural surface. The lungs are unusually solid and when removed from the body they completely maintain their shape. The lungs cut with great resistance and the knife becomes covered with black pigment. The nodules, which are usually seen in both lungs, may be present in greater number in one lung. Although nodules may be present from apex to base, the mid-lung field contains the largest number of nodules. Their average size is five millimeters to two centimeters. The silicotic foci stand out above the cut surface. Large emphysematous blebs are frequently observed in the apical and basal aspects of the lungs.

The uncomplicated form of silicosis has been designated by Simson, Strachan and Irvine[39] as "simple silicosis" in contrast to the "infective type," i.e., pulmonary silicosis and tuberculosis.

The gross appearance of the tracheobronchial and peribronchial lymph nodes in general is characterized by an extensive enlargement. They are firm, cut with great resistance and, on cross section, are of a slate-gray color. Although we have never observed such changes, Strachan[39] points out that periadenitis arising from chronic infection in the glands may cause cardiac embarrassment due to fibrosis around the blood vessels, and that oesophageal diverticulae are more common. Occasionally the root glands are small and soft.

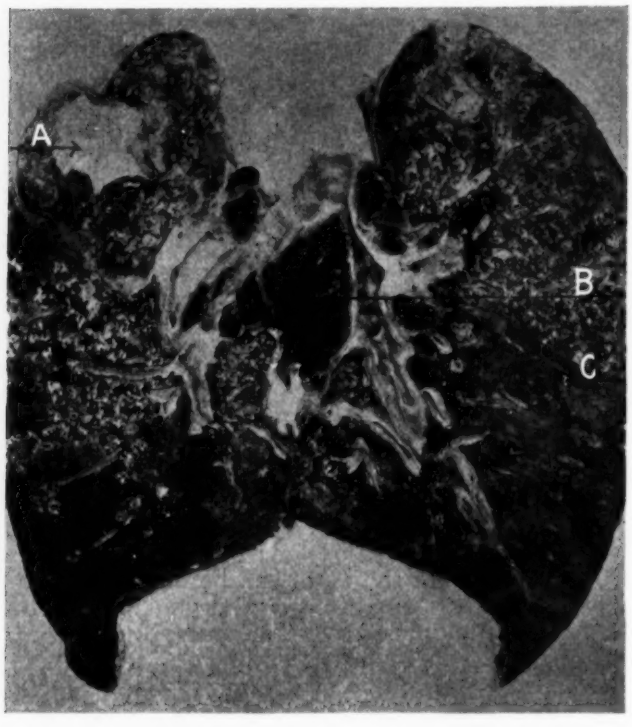

Fig. 6. Silico-tuberculosis (gross specimen). (A)—Large tuberculous excavation left upper lobe (note absence of trabeculation). (B)—Note the extensively enlarged silicotic tracheo-bronchial nodes. (C)—Silicotic nodules in lung parenchyma

Microscopic Appearance. The early changes observed in silicosis have been chiefly in animals exposed to silica. The initial reaction to dust is the production of large numbers of alveolar phagocytes which take up all of the dust particles reaching the alveolus. The alveolar phagocytes have been termed dust cells. Many of these pigment-laden cells escape through the air passages, while others are carried away by the lymphatics. In the inhalation of innocuous dust the rate of elimination follows the rate of invasion fairly closely, but in silicosis the rate of invasion far exceeds that of elimination. Mavrogordato stresses the fact that rate of elimination is more important than rate of invasion. As the inhalation of silica particles continues they are taken up by the lymphatic vessels and the cells migrate toward the pleura, in and beneath the surface of which they collect to form small nodules. This process has been designated by Mavrogordato[14] as "early pleural drift." Later the phagocytic cells are carried toward the tracheobronchial lymph nodes through the lymphatic vessels and many of them pass into the small lymphoid follicles lying along the course of the blood vessels and bronchi in the lungs. Many of the cells pass into the tracheobronchial lymph nodes. These aggregations of phagocytic cells in the lymphoid follicles have been called "pseudo tubercles." Fibroblasts and collagen fibrils soon appear between the phagocytic cells in the "pseudo tubercles."

As the process continues, the collagen fibrils become denser and phagocytic cells disappear; finally a hyalinized connective tissue area is present which takes on a whorl arrangement—it is the silicotic nodule which, in turn, is surrounded by a dense narrow zone of centrically arranged hyalinized connective tissue. The central zone contains a variable amount of black pigment—the extent of which deposition depends upon the amount of coal dust which is inhaled along with the silica. The black pigment may be so extensive as to overshadow the underlying connective tissue. These nodules become larger by the new arrival of dust cells at the periphery and there occurs a proliferation of fibrous tissue. The formation of the nodules compresses the lymphatic vessels and, in consequence of the ineffectiveness of the lymphatic drainage due to this blocking by dust cells in dense accumulation and due to pressure of silicotic nodules, the phagocytic cells pass right through the vessel walls into the surrounding vessel sheath. The presence of silica particles in these areas produces proliferation of connective tissue and this accounts for the thickened septa observed on gross examination in the early stages.

As the process continues, the individual nodules become larger and frequently border one another, but the identity of the individual nodule is retained. The septal connective tissue containing the vascular and bronchial structure becomes thicker as do the alveolar septa due to the accumulation of dust cells caused by faulty lymphatic drainage.

Microscopic Appearance of the Silicotic Lymph Nodes. In the early stages the nodes contain large numbers of phagocytic cells. Later collagen fibrils and fibroblasts appear between the cells the formation of the silicotic nodules being similar to this process in the lungs. Very little normal lymphoid tissue is seen in the late stages, when the nodules are in close proximity. Coal pigment is found within the nodules in greater amount than in the lungs.

Silicosis and Tuberculosis

TUBERCULOSIS is a frequent complication of silicosis. Irvine[39] and Gardner[7] believe that 75% of all silicotic individuals die of tuberculosis. Watkins-Pitchford[37] states that, after seven years of observation it was apparent that little more than half of the cases of simple silicosis seen in that time had developed overt tuberculosis changes, and many had died due to these changes.

	All Causes of Death	Tuberculosis	Pneumonia	Bronchitis
All Workers in General	790	142	66	38
Coal Miners	727	76	64	51
Tin Miners	1579	684	95	76
Lead Miners	1185	335	49	50
Iron Ore Miners	652	73	76	37
Sand Stone Workers	1427	415	120	116
Lime Stone Workers	753	129	54	38
Slate Quarry Workers	890	220	57	23

Table 1

The incidence of tuberculosis in silicosis varies according to the industries in which an individual has been employed. (Collis, Table 1). Ikert,[3] commenting upon the findings of Collis, believed that there is a higher incidence of tuberculosis in those silicotics where the silicic acid content is high.

An attempt to answer the question as to whether pneumoconiosis predisposes to tuberculosis has been made with animal experimentation. Cesa-Bianchi[40] infected with tubercle bacilli animals which had been previously exposed to dust and found that, while the controls not exposed to dust developed a mild form of tuberculosis, the animals previously exposed to dust died of an extensive pulmonary tuberculosis. Gardner found that tuberculous infection is facilitated when R^1 strain of tubercle bacilli is injected in animals previously exposed to dusts.

Jotten and Arnoldi[41] exposed cats to various dusts—steel filings, a hard and a soft porcelain dust, coal dust, soot and lime dust. The animals were exposed to the dust over a long period of time, after which they were infected with tubercle bacilli either aerogenously or subcutaneously, and then re-exposed to dust. In all examinations the

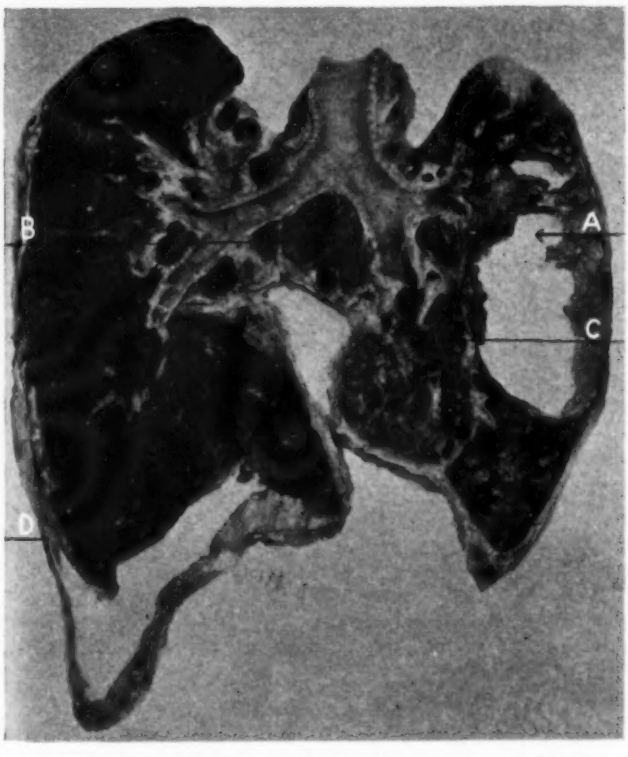

Fig. 7. Silico-tuberculosis—posterior view of lungs (gross specimen). (A)—There is a large tuberculous excavation which has ruptured through the interlobar septum. (B)—Note the markedly enlarged tracheo-bronchial nodes. (C) — In this specimen many silicotic nodules are seen in the lung parenchyma. (D)—There is extensive thickening of the pleura

animals exposed to the steel filings showed the most extensive tuberculous changes and these findings were attributed to the mechanical action of the dust. The severity of tuberculous changes in decreasing amounts occurred in the order of (1) hard porcelain, (2) soft porcelain dust and (3) coal dust or soot. Lime dust could not be found in the lung parts, but the tuberculous areas became calcified. The bronchial lymph nodes showed less evidence of tuberculous changes in those animals which were exposed to dust. The authors concluded that the previous dust inhalation had caused an obliteration of the lymph vessels and the tubercle bacilli could not be drained into the lymph nodes. These same authors observed that when animals were exposed to dust, later infected with mild doses of tubercle bacilli, and then re-exposed to dust, there was extensive new connective tissue formation. Furthermore, the caseous masses were encapsulated and the dust collections were frequently enclosed by dense connective tissue. Ickert[3] in commenting on these results believes

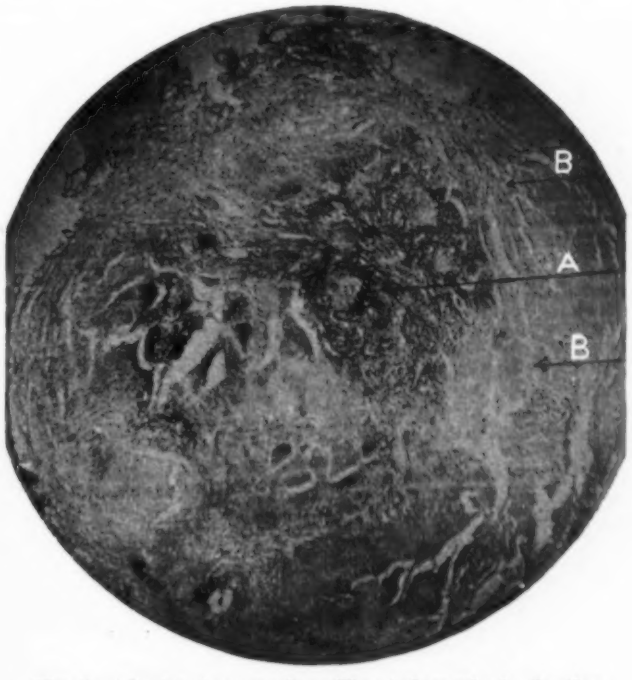

Fig. 8. Photomicrograph—silico-tuberculosis. In the center of the photograph there is a silicotic nodule showing the typical arrangement of fibrous tissue in whorls (A). In this silicotic area, coal pigment deposits are seen. Note the extensive caseous pneumonia around the silicotic nodule (B)

that in many cases of pneumoconiosis the indurated process is caused by the combined action of dust and tuberculosis.

The difficulty in accepting the findings of the animal experiments lies in the fact that tuberculosis in the human being is a late manifestation. Although Landis[15] believed that the tubercle bacilli may be inhaled with the silica particles. Watt and Irvine,[42] Cummins,[6] Alison[43] and Gardner[7] point out that in the majority of cases tuberculosis is a terminal phenomenon attacking the sufferer when the condition is already advanced, whereas in animal experimentation, the exposure to tubercle bacilli is soon after or during the early process of fibrosis.

Tuberculosis first manifests itself in silicosis at a much higher age (between 50 and 60) than it does in individuals not previously exposed to dust (between 20 and 30).

Views differ as to why tuberculosis so frequently develops in silicotic lungs. Beattie[44] believed that the reduction of effective lung tissue encouraged the lodgment of tubercle bacilli. Cummins[6] attributes the high mortality to the presence of inhaled tubercle bacilli in the fibrotic lung, where they must accumulate since the lymphatic drainage is blocked by the fibrosis, and whence they

exert a definite pathogenic and toxic effect upon their immediate vicinity. Gardner,[7] sets up against this view the fact that it offers no explanation of the phenomenon that when tubercle bacilli are injected intravenously in guinea pigs and rabbits, they select silicotic nodules for their site of development, growing better in silicotic soil than in normal animal tissue.

Gross Appearance of Silicosis and Tuberculosis. The picture will vary greatly with the extent of the tuberculosis found at autopsy. The lungs, although voluminous as in simple silicosis, are not as solid and do not keep their shape as well when removed from the body; the lung tissue has usually fallen together in the region of the cavities.

The basic tuberculous changes are the caseous

Fig. 9. Photomicrograph—silicotic nodule. Note the whorls of connective tissue with coal pigment deposition (A). This central area is surrounded by a concentrically arranged layer of hyalinized connective tissue (B)

pneumonic foci. These are found chiefly in the zones immediately surrounding the silicotic nodules. Frequently the gray, central silicotic focus is found to have fused peripherally with the yellow, caseous areas; the silicotic nodules do not, as in simple silicosis, stand out above the surrounding lung tissue but gradually fuse with it; the caseation also may extend inward into the silicotic nodule until the entire focus is transformed into a yellow area; and the more extensive the area of caseous pneumonia around the focus the greater is the difficulty experienced in distinguishing individual foci.

The larger areas of caseation undergo liquefaction resulting in cavity formation, and since this liquefaction takes place just beyond the silicotic nodules, the fibrotic foci tend to become loosened from their mooring and, in consequence, are expectorated along with the liquefied cheesy material. They are stony hard, gray, or gray-black, in consistency and color.

Tuberculous excavations, which are a fairly constant finding in silico-tuberculosis, and are present as a rule in the mid-portion of the upper and the upper parts of the lower lobe, are generally large and extend through the interlobar septum into the adjacent lobe, which rarely happens in uncomplicated cases of pulmonary tuberculosis. The trabeculae representing obliterated pulmonary arterial vessels, that traverse the cavity wall in simple pulmonary tuberculosis, are absent in silico-tuberculosis. Only a few cheesy particles are attached to the inner wall of the cavities and the inner margin of the excavation is often formed by silicotic foci. Only occasionally are the characteristic three layers of the cavity wall of uncomplicated pulmonary tuberculosis observed in pulmonary silico-tuberculosis. The reason is that in pulmonary tuberculosis, the cavity wall is formed by the response of the surrounding healthy lung tissue to the tuberculous infection. Since, in silicosis, the surrounding lung tissue is seldom healthy lung tissue but usually consists of silicotic nodules, an extensive productive reaction is impossible. However, when healthy lung tissue does surround the liquefying area of caseous pneumonia, a dirty-gray membrane forms the inner wall of the cavity. Beyond this, there is a narrow red zone with an adjacent outermost layer formed

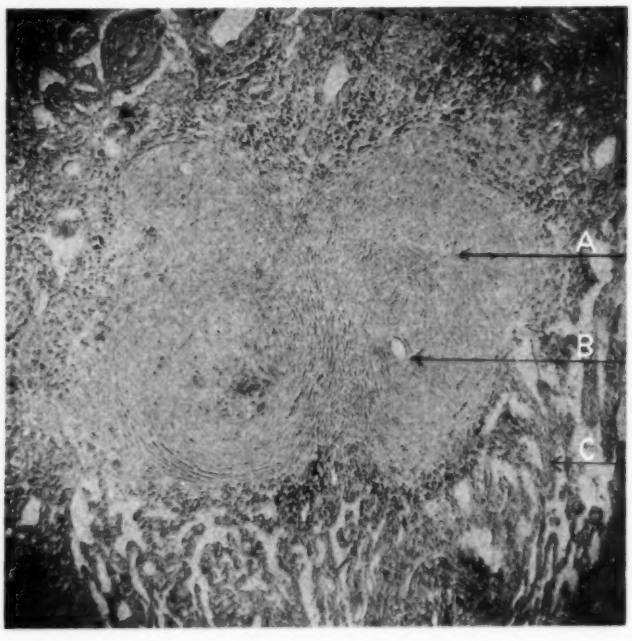

Fig. 10. Photomicrograph — silicotic nodule. This specimen shows the nodule to be composed of hyalinized connective tissue arranged in whorls (A). Note the diminution of lumen of small blood vessel (B). The alveolar septa are markedly thickened (C)

by firm silicotic nodules. The tuberculous excavation in a number of places ruptures into the pleural space.

The tracheobronchial and peribronchial lymph nodes generally show no gross evidence of tuberculous change.

The pleurae are much thicker than in simple silicosis, and tuberculous pleuritis is a fairly frequent late complication due to the rupture of a tuberculous excavation into the pleural space. The emptying into the pleural space of cheesy particles containing tubercle bacilli gives rise to tuberculous pleuritis, with which the visceral surface of the parietal pleura and the parietal surface of the visceral pleura become covered with a densely fibrinous deposit and soft cheesy particles.

Microscopic Examination. We cannot share the general opinion of Beattie,[44] Ickert,[3] Gardner[7] and others that, in silicotics, pulmonary tuberculosis assumes a fibrotic character. Ickert is of the opinion that, inasmuch as tuberculosis in pneumoconiosis is predominantly productive in character, there is justification for speaking of "a special type of pulmonary tuberculosis" and he gives it the name of "Staublungentuberkulose." We have found from studies that tuberculous changes first begin in the alveolar spaces and that the process is predominantly exudative in character.

The basic tuberculous changes that we have observed in silico-tuberculosis indicate that it is a caseous pneumonic process. At first, the alveoli immediately surrounding the silicotic nodules are

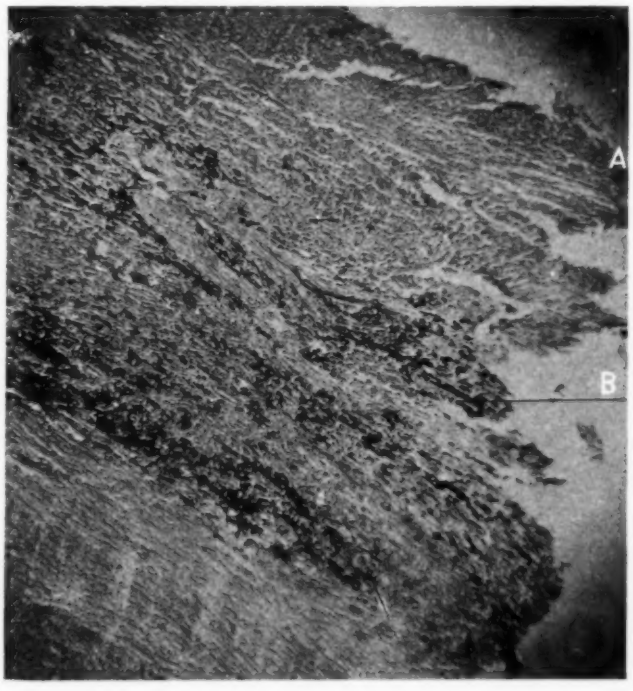

Fig. 11. Photomicrograph.—.cavity wall in silico-tuberculosis. The specimen shows part of a tuberculous cavity. The inner wall of the cavity is lined by caseous material (A). Beyond the caseation is a fibrous nodule with marked coal pigment deposition (silicosis)—B

filled with an exudate composed of fibrin, polymorphonuclear leucocytes, alveolar phagocytes, lymphocytes and red blood cells. As the process continues the cellular elements undergo necrosis and the fibrin becomes swollen until end-stage caseation is reached. The caseation is pink and granular and, within it, one can still frequently discern outlines of alveoli. The areas of caseous pneumonia may completely encircle the silicotic nodules. The alveoli surrounding the caseous areas contain a minimal exudate composed of serum, alveolar phagocytes, lymphocytes and red blood cells (perifocal inflammation). If there are no alveoli available in which it may develop, perifocal inflammation is absent, and the silicotic nodules border the areas of caseation. The caseous pneumonic foci may become encapsulated or may undergo liquefaction: the smaller the area the greater is the opportunity for it to undergo encapsulation. There is a productive reaction at the periphery of the caseation in the form of fibroblasts, epithelioid cells, and giant cells. In the later stages collagen fibrils laid down by the epithelioid cells increase in size and thickness and with this there is a decrease in the number of cells until finally a hyalinized connective tissue capsule is formed.

Should the area of caseation extend into the silicotic nodule, the original area of caseation is often still distinguishable from the necrotic silicotic focus. The silicotic nodule is granular in appearance and individual fibers within it are no longer clearly discernible. In silicosis which is productive in character elastic fibers are destroyed, whereas in the caseous pneumonic focus the elastic fibers of the alveolar septa are clearly and easily discernible with the elastica Van Gieson stain.

Cavity formation in silicotic lungs does not differ from that in simple tuberculosis. The area of caseous pneumonia is invaded by dense collections of polymorphonuclear leucocytes which liberate proteolytic enzymes. These enzymes digest the caseous material resulting in the formation of a gelatinous substance which is expectorated. The completed cavity wall is composed of an inner layer of thick bands of fibrin containing polymorphonuclear leucocytes, fibroblasts and

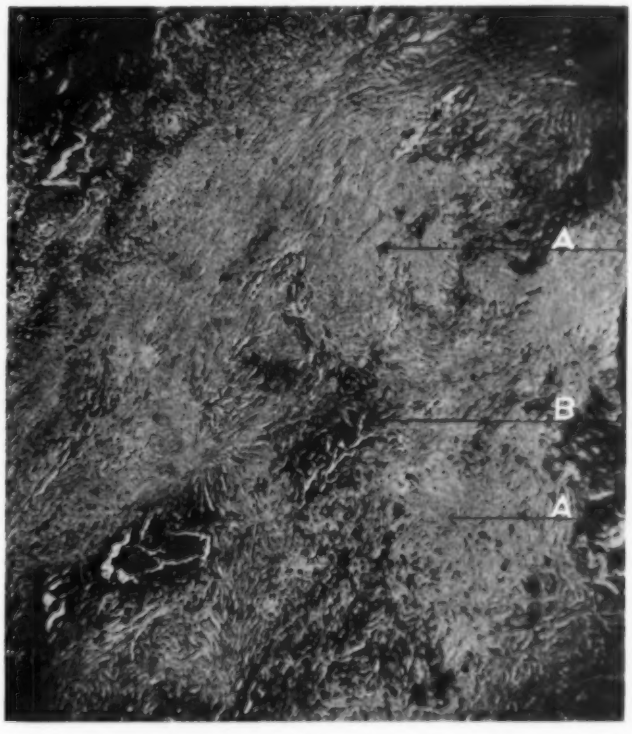

Fig. 12. Photomicrograph—silicosis—tracheo bronchial lymph node. Note the extensive fibrosis which has completely replaced the normal lymphoid structure (A). There is extensive coal pigment deposition (B)

lymphocytes. This layer has been designated the pyogenic membrane. Next to this is a zone of vascular granulation tissue, and the third layer of the wall of the cavity infrequently observed in silicosis is a zone of fibrous tissue containing compressed alveoli. This layer arises from the organization of the perifocal inflammation around the original caseous pneumonia. Occasionally, in silicosis, the cavity wall is composed of only an inner pyogenic membrane bordering on a silicotic zone. In other instances, there are present two inner zones of the wall with silicotic foci forming the outer wall. The extent of the formation of

the outer two layers depends upon the amount of uninvolved lung tissue intervening between the original caseous pneumonic foci and the peripheral silicotic nodules.

The silicotic nodules expectorated in the sputum often show no microscopic evidence of caseation. The whorl arrangement of the fibers is clearly seen and the nodules are laden with black pigment.

Miliary nodules also are present in the lung tissue, usually in the form of an epithelioid giant cell tubercle.

Microscopic Appearance of the Tracheobronchial Lymph Nodes. The tuberculous changes in the lymph nodes are much less extensive than in the lung. Since the involvement of the node depends chiefly upon the lymphatic drainage of tubercle bacilli from the tuberculous foci, it is understandable that few bacilli get into the lymph nodes—the extensive blocking of the lymphatic

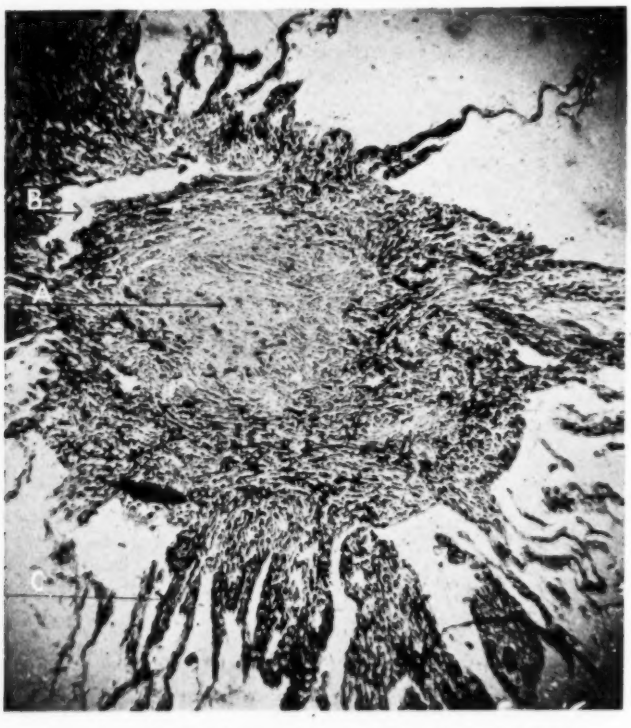

Fig. 13. Photomicrograph — silicotic nodule surrounding a blood vessel. The silicotic nodule with its densely arranged hyalinized connective tissue (A), surrounds the small blood vessel (B). The alveolar septa surrounding the focus are markedly thickened (C)

system accounts for this fact. The lymph nodes usually contain small areas of caseation, mainly in regions previously uninvolved by the silicotic process, surrounded by epithelioid cells, giant cells and fibroblasts. Frequently the areas of caseation extend to involve the neighboring silicotic areas. Small epithelioid-giant cell tubercles are seen in the node. The greater part of the node however, is composed of intact silicotic nodules.

Although silicotic nodules have been described as occurring in the liver and spleen, we have never observed them. The occurrence of these foci in such organs can take place only when there has been a blood stream dissemination of silica particles. These particles are generally collected, however, in the tracheobronchial nodes and the lymphatic vessels are blocked, so that lymphatic drainage into the blood stream is practically impossible. The microscopic and gross appearance of the silicotic nodules is no different from those in the lung and tracheobronchial lymph nodes.

Silicosis is frequently observed in the mediastinal and peripancreatic lymph nodes, due to the fact that they are intimately connected through the lymphatic vessels with the tracheobronchial lymph nodes.

Asbestosis

THE knowledge of asbestosis has developed in the last 15 years through the studies of Cooke,[45] McDonald,[46] Oliver,[47] Simson[48] and Stewart.[49]

Pulmonary asbestosis has occurred in individuals who handle and crush crude asbestos, make asbestos mattresses, or card and weave fine asbestos. As many women are employed in this work, more women than men are exposed to this dangerous dust.

Stewart is of the opinion that asbestosis is definitely a silicatosis, and that the fibrous mineral, asbestos, is of a variable chemical composition

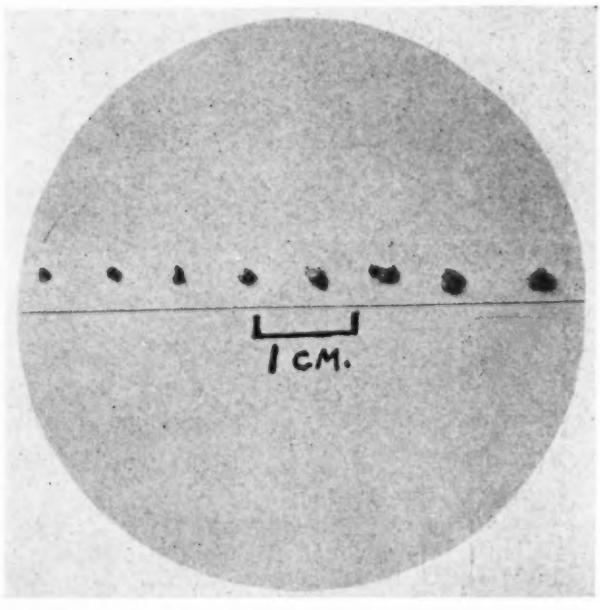

Fig. 14. Hard masses found in sputum of an individual who died of silico-tuberculosis (From a case of Dr. Paul Slavin)

according to its source. The silica is usually combined with magnesium and iron.

The length of exposure required to develop asbestosis is shorter than for silicosis, varying from four to 13 years although Wood[50] observed two cases develop after an exposure of four and 15 months respectively. The disease is a steadily progressive one and is fatal within a shorter period than silicosis (Stewart). On the other hand Lanza[52] has found that in the United States the course of asbestosis is a much milder one than that reported by the English (Stewart,[53] Wood[50] and others), although he believes that it is possible that English factories may be more dusty than those in the United States. The complication of tuberculosis in asbestosis is believed to occur much less frequently than in silicosis.

Gross Appearance of Asbestosis. We have never observed a case of asbestosis at postmortem in Sea View Hospital, but a comprehensive study of

both gross and microscopic specimens has been made by Stewart.[53] The pulmonary fibrosis is diffuse in character, peribronchial and basal in location, in contrast to silicosis where the fibrosis is nodular in character and present in the upper part of the lungs. The fibrous areas early in the disease are gray in character; later, with the faulty elimination of coal pigment that results from the blocking of the lymphatics, the areas turn to gray-black. As the process continues, the fibrosis extends into almost all portions of the lung tissue. Bronchiolectasis and bronchiectasis are present within the substance of the fibroid areas, and bronchopneumonia and acute tracheobronchitis often cause the death of these patients. The pleurae, especially in the basal regions, are thickened and adherent. In some cases the pleural sac is completely obliterated; in others a fibrinous or serous exudate may be present within it.

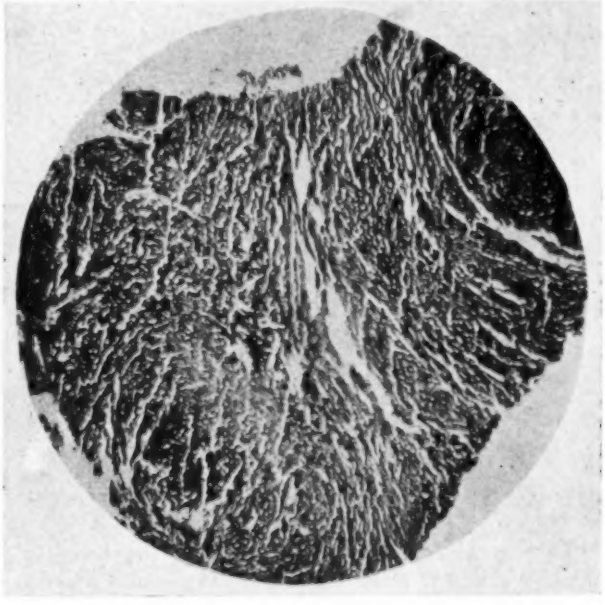

Fig. 15. Microscopic picture of one of these firm masses showing a whorl arrangement of connective tissue with coal pigment deposition

Microscopic Appearance of Asbestosis. In the early phases of the disease there is a thickening of the alveolar septa as a result of fibroblastic proliferation. The alveolar spaces contain numerous alveolar phagocytes. With progression of the disease, fibrosis becomes more marked, the alveolar structure gradually disappears and, in its place, there is now dense fibrous tissue containing coal pigment in variable amounts. The few remaining alveoli which lie in the area of fibrous tissue are lined by a low cuboidal epithelium and have a glandular appearance.

Scattered throughout the lung in both the diseased and healthy parts are spindle-shaped structures (first described by McDonald[46]) known as asbestosis bodies. These bodies are slender in their center and bulbous at each extremity. Many are arranged like strings of graduated beads with the largest at the end of the chain. Other bodies are unsegmented. In the unstained state they range from yellow-green to deep brown; when stained with potassium ferrocyanide they give a Prussian blue reaction.

These asbestosis bodies are found in the sputum of individuals after the first two years of exposure to asbestos dust (Lynch,[54] Stewart,[55] Strachan[56]). Their presence, however, is not an indication that the pulmonary fibrosis is present, but that asbestos dust has been inhaled and has remained in the lung tissue for some time. Stewart, Tattersall and Haddow[57] point out that the significant findings in the sputum are clumps of asbestosis bodies. The finding of these bodies points to disintegration of lung tissue.

Siderosis

THIS type of pneumoconiosis is only infrequently encountered. Bohrad[58] who reviewed the literature from 1866 to 1930 found that only 30 cases of siderosis were reported. The disease developed in individuals who have been exposed to hard iron ore dust over a long period of time. Cronin[59] and Stewart[60] believe that the most dangerous occupation is the use of the dry drill which generates a large quantity of dry dust which is shot straight onto the worker. Stewart and Faulds point out that before the use of the dry drill in the Cumberland mines of England, iron ore mining was considered a healthy occupation.

The silica content of the iron ore is from 12 to 13% (Cronin,[59] Stewart and Faulds[60]). Chemical analysis of the lungs has revealed the presence of large quantities of hematite (iron dust) in the lungs and at least as much silica as in the ordinary cases of silicosis. Carleton[61] found that exposing guinea pigs to hematite dust was comparatively harmless and that it stimulated phagocytosis and was rapidly removed from the lung. Stewart and Faulds[60], Goadby[62] believe that the silica is the real cause of the fibrosis and that the lesion is really one of silicosis, and they have applied the term sidero-silicosis to this disease. The hematite, according to Cummins and Sladden,[63] accumulates in the fibrotic area just as coal dust does in the silicotic lungs.

The gross and microscopic picture of pulmonary siderosis is for the most part similar to silicosis. The gross picture differs from silicosis in its color. In siderosis the lungs are brick-red, dark-red or chocolate-brown in appearance. In simple silicosis the pulmonary fibrosis is present chiefly in nodular formation, whereas in siderosis the massive or the diffuse type is most often observed. The nodular form is only occasionally found.

Anthracosis

THE term anthracosis was first suggested by Stratton in 1838. Animal experimentation and studies of human lungs have led to the conclusive view that coal dust does not lead to fibrosis unless it is inhaled along with silica dust.

Willis examining the lungs of guinea pigs exposed to coal dust for a period of 10 months found only slight coal pigment deposition in the lungs and no evidence of fibrosis. Similar observations were made by Mavrogordato,[14] Henrius and Richert.[64]

Mavrogordato[14] has shown in the study of the lungs of guinea pigs exposed to various dusts that the initial response to the particles which produce fibrosis (silica) and those which do not produce fibrosis (coal pigment) is the same. Large numbers of alveolar phagocytes are produced and appear in the alveolar spaces to take up the dust particles which have entered. In anthracosis the life of the dust cells (alveolar phagocytes) is a

brief one in contrast to silicosis where the dust cells are preserved for a long time by the silica. The cells are either carried off by the lymphatics, or they die and undergo autolysis, or they are digested by the tissue juices. The dust cells are carried to the perivascular lymphoid zones in the lungs. The dust cells in these areas become disintegrated so that weeks after exposure the dust lies extracellularly on the outer surface of the blood vessels.

The presence of gross fibrosis in the lungs of miners is due to a definite accompanying or antecedent silicosis and fully justifies the term silico-anthracosis (Cook,[65] Oliver[66]). It is generally agreed that the presence of pulmonary tuberculosis in coal miners (silico-anthracosis) is not a frequent occurrence. (Table 1). Cummins[6] is of the opinion that the reason is that the finely divided coal dust is not uniformly distributed but lies in great abundance at the apices, in the subpleural regions and in the peribronchial and perivascular lymph channels. This accumulation is the result of lymphatic blockage in these fibrotic lungs. This author believes that coal dust is able to remove a considerable amount of active element tuberculin, and thus has the effect of detoxifying the tuberculous foci which must, he believes, arise in a silicotic lung from time to time.

(I am indebted to Dr. Hyman Alexander and Dr. Samuel Lipstein for their aid in the preparation of this article.)

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Minor Surgery

Book Review by

C. O. Sappington, M.D., Dr. P.H.

Christopher's "Minor Surgery" has always been an excellent guide, not alone for the surgeons, but also for the general practitioner. This Third Edition, published in 1936, by Frederick Christopher, M.D., F.A.C.S., Associate Professor of Surgery at the Northwestern University Medical School, Chicago, with the foreword by Allen B. Kanavel, M.D., F.A.C.S., Professor of Surgery at Northwestern University Medical School, contains some very new material and interesting facts.

According to the preface to this edition, new material deals with new methods for promoting wound healing, bacteriophage, resuscitation upon the operating table, de Takat's method of ambulatory vein ligation, effort thrombosis, leech treatment of phlebitis, x-ray treatment of gas gangrene, bismuth injection treatment of warts, sodium morrhuate in cystic hygroma, "sprinter's fracture," lymphogranuloma inguinale, Elliott treatment of pelvic inflammatory disease, torus fractures, glomus tumors, iliopectineal bursitis, Leriche treatment of sprains, aspiration biopsy, Wangensteen stomach suction apparatus, injection treatment of hernia, and various others.

The work is replete with interesting and explanatory diagrams, photographs, and x-ray reproductions, all of which help to explain the technical details and statements made in the text. It is a book of inestimable value to the general practitioner, to the industrial physician and surgeon, and to anyone who engages in practice.

The volume contains 1030 pages, with 709 illustrations, is priced at $10.00 in cloth, and is published by W. B. Saunders Company, Philadelphia and London.

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Vital Economy

"POLITICAL economy and social economy are amusing intellectual games; but vital economy is the philosopher's stone." This is from the brilliant and prolific pen of George Bernard Shaw. It is one of those statements so well made that the reader is rather apt to think less of what is said than of the manner of saying it.

The analytical mind, however, will seize upon "vital economy" as having the significance—even while some question may exist as to what it is.

Should the interest be pursued, from the point of view of the medical man in public health work the subject will be found apparently more concerned with vital statistics than with vital economy—"apparently" because the effectiveness of the one is measured in terms of the other, and the yardstick is in constant and obvious use.

But from the point of view of the industrial physician and surgeon who has given thought to the field of practice in which he is engaged, there is a very clear conception of the term—for is it not a name for his field? Because he is in that field his work is vital not only as a matter of life, but also, and equally, as a matter of labor. And therein is the economy, for in any interdependence—and where is the community of which interdependence is not the life blood—what does a man possess except his ability to do his share, in one way or another, toward the permanency and peace and profit of that interdependence?

Fundamental in all of these is labor, work, the ability to do. And compensation for enforced inability to do, arising from and out of the circumstances of the doing, is not a substitute, but only an unsatisfactory palliative. Compensation, however, unlike palliatives in general, is permanent. As to injuries it is already integral with the industrial structure; as to occupational diseases it is being worked into a key place in the design. It will remain permanent so long as there is a need for it. And the need will persist until it is eradicated by prevention.

The work of the medical men in industrial affiliations has been said to be "mostly curative; hardly at all preventive." Once in a while this is spoken critically, as if the medical men were themselves to blame. Possibly they are, in some respects. But for reasons! There is drama in the emergency of injury; while prevention on the whole—and particularly of occupational diseases—is drab, routine, unvivid. Prevention can be machanical only up to a certain point; from there on, it is an individual matter—human. And the fact that it is human impresses it with an appalling inertia. This inertia is very difficult to overcome, and, because employers are employers, has only one sure approach to its overcoming. That is by way of *cost*. When the relation of lack of prevention to cost is made clear to the employer, then—but, sad to say, rarely until then—he begins to think along preventive lines.

It is not the medical man's fault that this is so. Indeed, it is not anyone's fault in particular—because the matter of the cost of things is inseparable from the mass of our whole scheme of living. But it may be the medical man's fault that the employers who pay are not more fully aware of how much they are paying, what they are paying it for, and how much less they would be compelled to pay in some respects if they would voluntarily pay a little more in others. It may even be the medical man's fault that he himself doesn't know, and so is not in a position to tell the employer.

The statement of this probability—or is it the actual condition?—suggests the remedy. Study, in terms of cost to the employer *as such*, all the conditions that come to the medical department from employees *as such*. What are their particularized as well as their aggregate figures? Then dramatize them—how much more of customer's goods would the aggregate pay for? What additional percentage would it make available for dividends? With the figures known, and with some dramatization in their presentation, the still necessary job of selling preventive medicine to the employer as a matteer of vital economy—vital to the humans in his employ, vital to his own expense account, and therefore a measure of economy to both—will be made much easier.

Letters

Medical Department Costs

TO THE EDITORS OF INDUSTRIAL MEDICINE:

WE ARE now entering a period of renewed industrial activity, and industrial diseases, along with other elements in industrial relationships, are already receiving new and added emphasis. As a result of this I believe that more and more industrial concerns are going to start Medical Departments or enlarge the ones

they have. If my belief is sound, we should be in a position to talk intelligently regarding the costs of proper Medical Departments, not only on the basis of an individual instance, or an aggregate, but also on the basis of comparative costs as between one kind of business and another.

Certain figures are to be had, of course, but these are not available in any way that will permit of comparisons; they can not be related one to another *per item,* as to the elements of cost. We need the means of comparison. It is disconcerting to the executives of an industry who are considering the establishment, or even the enlargement, of a Medical Department to have quoted to them figures varying from $3.00 to $10.00 per employee per year, when they know, but cannot find out why, the services rendered in some plants which have Medical Departments are comparable in everything but cost.

The American College of Surgeons has given us a yardstick for standards of service; we ourselves ought to be able to state that the maintenance of that standard under any given conditions will cost just so much.

I am, therefore, making the suggestion that INDUSTRIAL MEDICINE take it upon itself to see if we can get the various industries which have Medical Departments to make their figures available to us on a standard form, arranged for comparative analysis. And I submit the accompanying outline as such a form, and propose that you conduct a questionnaire survey, and tabulate the replies so as to make their figures available to executives and medical men seeking accurate information on this subject.

—C. F. N. SCHRAM, *Medical Director, Tennessee Eastman Corporation, Kingsport, Tennessee.*

QUESTIONNAIRE
PLANT MEDICAL EXPENSE

Please Mail to
INDUSTRIAL MEDICINE
844 Rush Street,
Chicago

This part of the form is a working copy.

Item	Total Cost	To Plant Medical	To Compensation	To Welfare	Cost per employee: Plant Medical	Compensation	Welfare
1. Doctors' Salaries							
2. Nurses' Salaries							
3. Clerical Force							
4. Outside Doctors, Nurses, and Fees							
5. Hospital Expense							
6. Legal Expense							
7. Medical Supplies							
8. Building and Equipment Costs							
9. Heat, Light, Power							
10. Traveling Expense							
11. Miscellaneous							
12.							
TOTAL							

Instructions: Please show the total cost of operating your plant Medical Department, breaking it down as per the form shown on the reverse side.

Name of plant _____

Street _____

City and State _____

Signed _____

MEDICAL DEPARTMENT CHARGES:

That part of physicians' and nurses' time spent on physical examinations, preventive medicine, minor (not lost time) injuries, plant inspections, visiting and other work not connected with lost time (Workmen's Compensation) injuries. This is a variable amount, but an average can be computed.

All medicine or drugs used in preventive medical service.

All bandages, medicine or drugs, and splints used on minor injuries and lost time (Workmen's Compensation) injuries at the Medical Department or First Aid Station in industry.

Upkeep and other fixed or incidental expenses of the Medical Department such as maintenance and repairs, power, water, insurance, supervision, depreciation and taxes.

WORKMEN'S COMPENSATION CHARGES:

That part of the industrial physician's or nurse's time spent on the care of Workmen's Compensation patients.

All consultation fees and the expenses attendant thereto on Workmen's Compensation cases.

Compensable injuries cared for by private practitioners (outside doctors).

All medical testimony regarding Workmen's Compensation claims.

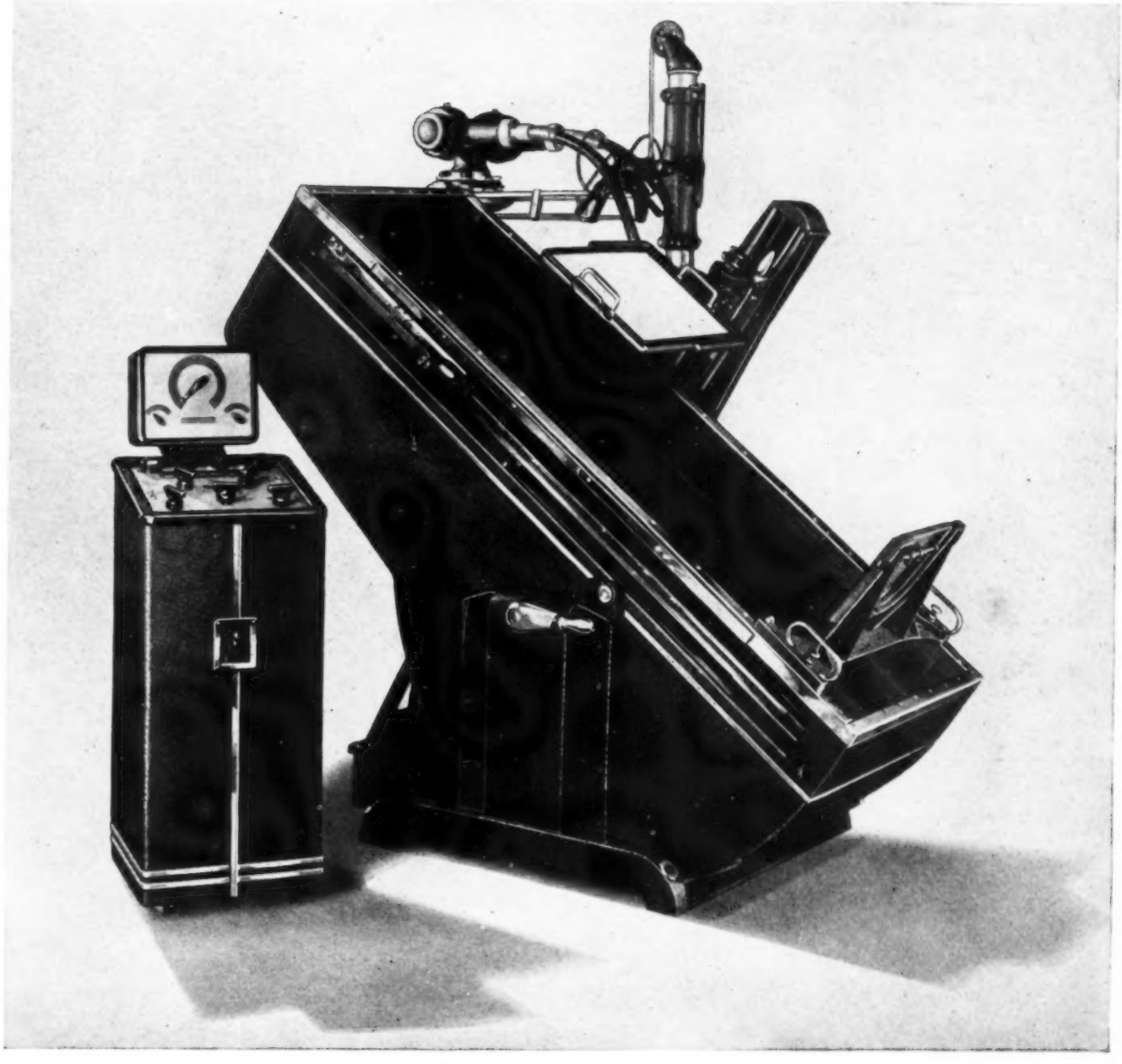
Medical examinations to estimate disability.

All legal fees regarding Workmen's Compensation claims and all other Workmen's Compensation expenses.

All hospitalization of Workmen's Compensation patients.

WELFARE CHARGES:

All outside expenses incurred by the industrial organization in the diagnostic, paliative or curative care of non-compensable injuries or illnesses and which are undertaken because of long service or from other purely welfare motives.



No matter what the yardstick

More accurate diagnosis, more certain prognosis, shorter disability time, lessened burden—it matters little what the yardstick might be, the final result is the same. In any event, the routine use of x-ray in the industrial clinic has fully proved its practical value—proved it in the accumulated experience of more than a hundred plants using Westinghouse X-Ray equipment. We particularly suggest that you let us give you some facts about the Fluoradex, the compact, complete diagnostic unit, for its many models cover so wide a range of capacity and usefulness that it is indeed ideally suited to industrial requirements—from first aid room to the large hospital.

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Industrial Medicine and Surgery

Surgical Shock from Burns, Freezing, and Similar Traumatic Agents

THE various signs and symptoms that, combined, give the picture of surgical or traumatic shock are too well known to be elaborated at this time.* It is of interest, however, to consider the enormous amount of speculation concerning the causes of shock. The picture is striking of a severely injured patient with full consciousness and often no external hemorrhage gradually sinking into a collapsed state with cold, clammy skin, fast thready pulse and greatly lowered blood pressure and later coma and ultimately death intervening. Such a syndrome has naturally piqued the interest of numerous clinicians, and some of them have carried this interest into the experimental laboratory. Their observations led to two main standard theories as to the origin of shock. The nervous theory embraced nervous trauma, vasomotor collapse, noci-association and other concepts. The toxic theory involved the supposition that traumatized tissue gave rise to toxic products. But since shock could be produced experimentally in the absence of nervous connections to the traumatized region on the one hand and, on the other, extracts of traumatized tissue could be injected into other animals without ill effects, both of these theories remained unproved.

Recently a third theory has come to the fore, chiefly as a result of the simultaneous work of Blalock[1] and Phemister[2]. This theory involved the demonstration that in many cases of shock there is hidden extravasation of blood in the traumatized tissues in amounts hitherto unrealized. Experimentally these authors showed that if one hind limb of an anesthetized animal is severely traumatized, and then by weighing the hindquarter sections the weight of the traumatized and opposite normal limb is compared, the swelling due to hemorrhage into the traumatized limb may be large enough to correspond to a loss of three liters of blood in an average-sized man. The writer has made measurements of limbs and other injured regions of patients admitted in shock on the traumatic service of Billings Hospital. These injured regions were often swollen very soon after the injury, ecchymoses being visible, sometimes blood being aspirated from large hematomas and the amount of swelling was in many instances far greater than would have been realized had not direct measurements been made.

The advocates of the local blood loss theory of shock do not maintain that this is the only factor. The loss of large amounts of blood into injured areas is, however, just as serious as the more evident external hemorrhage. From the standpoint of therapy this concept is important because it is easier to treat a patient who is suffering from loss of blood than one who is affected by some vague ailment such as noci-association. Blood transfusion has then a more definite place in the treatment of shock. If blood is not immediately available its place may be temporarily taken by blood substitutes such as intravenous saline, Ringer's solution or glucose. If blood has been lost, however, blood should be restored and our experience at the Billings Hospital has borne out this fact in the treatment of shock cases.

Work done by various workers has shown that a local blood or blood plasma loss may explain collapse of blood pressure in other shock-like conditions such as pulmonary edema, intestinal trauma, mesenteric thrombosis, burns and freezing. All of these conditions represent injuries due to trauma whether that trauma be thermal, chemical, or mechanical. It is chiefly with the effects of thermal trauma, both as regards burns and freezing that the present paper has to deal.

Burns

DAVIDSON[3] originally introduced the tannic acid treatment in the belief that tannic acid would precipitate toxic proteins formed in the burned tissues and prevent their absorption. Some more recent work especially by Underhill[4], Blalock[5], and the writer[6] indicated that there might not be any burn toxin acting at all, but that death in burns might be due to loss of blood plasma from the blood stream in enormous amounts. The extent of this plasma loss may equal 60% of the plasma normally present in the circulating blood stream. Not only is this fluid lost into the burned tissues, but it leaks out from the weeping burned surface. All of us who have used the tannic acid treatment have noted a white precipitate formed when the tannic acid strikes such a weeping surface. This white precipitate, not unlike that formed when urine is tested for albumin, serves as a qualitative test for protein in a burn exudate. In several of our cases a quantitative test for protein in the blister fluid showed that it is present in the same concentration as in blood plasma. It stands to reason that the loss of this plasma-like fluid either into the burned tissues or from the burned skin is of much more serious consequence than the loss of a mere watery transudate. As an example in kind, it is obvious to all of us that while one can urinate two to three liters a day, the loss of two to three liters of blood is of quite serious import.

Whether one subscribes to the toxic or the fluid-loss theory of causation of secondary shock in burns, two facts stand out: first, that secondary shock is one of the chief causes of death from burns, and second, that tannic acid seems to be effective in combating such shock when present. It matters little whether the tannic acid acts by precipitating toxins or by sealing off the capillaries and preventing fluid loss, the important thing is that to the best of our knowledge at the present time, tannic acid combats shock and lowers the death rate in burns.

Before the introduction of tannic acid by Davidson[3] in 1925 burns were treated with a variety of occlusive dressings, paraffine, cod liver oil, carron oil, sodium bicarbonate, picric acid, saline dressings, horse serum, cresol and Dakin's solution. Since 1925 a number of protein precipitant methods have been evolved, most of which depend on tannic acid for their action. These are briefly outlined in Table I.

A. Tannic Acid Preparations—
1. Tea (Chinese, 5000 B.C.).
2. Ink treatment (Jews and Filipinos. Older inks contained considerable tannic and gallic acids, modern inks sometimes are chiefly coal tar products).
3. 2.5% solution (Davidson, 1925).
4. 5% solution (Used mostly in U.S.A. at present).
5. 20% solution (Used in England, Wilson, 1928).
6. Tannic acid baths (Wells, 1933).
7. Tannic acid buffered with sodium bicarbonate (Seeger, 1932).
8. Cutch extract (60% tannin).
9. Tannic acid plus silver nitrate (produces eschar more rapidly than tannic acid alone, Bettman, 1935).
10. Tannic acid jelly.

B. Tannic Acid Plus Antiseptic Base—
1. Acriflavine, 1/1000 (Wilson, 1934).
2. Dettol, 20% (Clark and Cruickshank, 1933).
3. Bichloride of mercury (1/2000) (Mitchiner, 1933).

C. Gentian Violet, 1% (said to combine antiseptic properties especially against streptococci with the production of a more pliable eschar than tannic acid, Aldrich and Firor, 1933).

D. Ferric Chloride, 5% (Eschar more rapidly formed than by tannic acid and more pliable, Coan, 1935).

Table 1. Protein Precipitant Methods of Burn Treatment

As Seeger[7] has pointed out, the ideal site for the use of tannic acid is on large areas of skin on the trunk and thighs. The spray should be replaced by a water-soluble tannic acid jelly in burns about the face and eyes. Care should be taken in burns that completely encircle the lower leg and forearm and particularly the fingers to avoid constriction from a firm encircling band produced by the eschar. Butesine picrate ointment may be preferable on these areas.

* HENRY N. HARKINS, M.D., PH.D., Chicago; from the Douglas Smith Foundation and the Department of Surgery, the University of Chicago; Delivered at the Sixty-sixth Annual Session of the Colorado State Medical Society, September 12, 1936, at Glenwood Springs; Published in *Colorado Medicine*, December, 1936.

Tannic acid should not be used after too long an interval has elapsed from the time of burning.

It must not be lost sight of at this point, however, that in severe cases shock is often present when the patient is first seen. Tannic acid merely helps prevent to some extent at least further loss of fluid and to no degree restores what is already lost. Other general methods of shock treatment such as morphine, heat, fluids and most important of all, blood transfusion are to be applied as soon as the patient is seen.

Blood Concentration in Severe Burns: An oft-observed clinical fact that has had repeated laboratory verification is the presence of marked blood concentration in extensive burns. Tappeiner[8] in 1881 was the first to report this finding. One of his patients had a red count of nine million following a severe burn. Locke[9] in 1902 reported four cases of burns with a red count of nine million or more. Underhill[4] in 1923 reported hemoglobin percentages as high as 209. McClure and Allen[10], at the Henry Ford Hospital, state that "a blood concentration of 125% is indicative of a dangerous condition and one of 140% is a condition which is not for long compatible with life." The presence of blood concentration is often a better gauge of a severely burned patient's condition than is the blood pressure. The writer has shown experimentally[6] that the blood concentration is proportionate to the plasma leakage, but that the blood pressure may remain compensated until near the end, only to collapse suddenly just before death. Any severely burned patient should have careful hemoglobin and red count determinations during at least the first 48 hours following the burn.

Freezing

CLINICAL cases of freezing differ markedly from burns in several very important respects. A patient may be burned over a certain portion of the body surface without his general body environment and temperature being affected to any marked degree.

On the other hand, freezing is induced slowly, and it is hard to conceive of how one could freeze a large portion of the body without being generally chilled. Minor frostbites of fingers, ears, the nose, and even the hands may occur while the body as a whole does not suffer, but to freeze a larger region there is almost invariably an associated depression of general body temperature. Furthermore, while burn cases usually occur in civilized centers where medical attention is at hand, freezing most often occurs in arctic or isolated regions.

The writer began a study of freezing with two purposes in mind. First, to test the time honored and possibly empiric practice of gradual thawing frozen extremities; and second, to see if in freezing as in burns there is a shock-like condition with lowered blood pressure, and if so, to determine if its mode of production might not be the same.

Possible Dangers of Rapid Thawing: The universal practice of gradually thawing a frozen member, as by the application of snow, has not been thoroughly tested experimentally. Maximow[11] states that in plant cells it is the freezing and not the rapid thawing that causes the harm. Nagelsbach[12] states, however, that he finds neither clinical nor experimental support for the practice of gradual thawing and that, during the World War, the surgeons who adopted the practice of heating frozen limbs as rapidly as possible had at least no poorer results than the others.

Experiments: Both hind limbs of thoroughly anesthetized dogs were frozen solid by the application of solid carbon dioxide. Then one limb was placed in water at 42° C. and the other in water at 12° C. and after thawing, no difference was found in the subsequent behavior of the limbs over a period of 18 hours.

Both ears of thoroughly anesthetized rabbits were similarly frozen. The amount of freezing was enough so that when thawing was done at room temperature about one-third of the ears would become gangrenous. In a series of eight rabbits in which one ear was thawed at 38° C. and the other at 2° C. over a period of 10 minutes and the ears observed over a period of several weeks, the amount of swelling and subsequent gangrene was essentially similar on the two sides. If anything, the ears that were thawed in ice water showed more gangrene.

Shock and Plasma Loss in Freezing: Solid carbon dioxide was applied to about one-fourth of the total body surface on one side of 10 completely anesthetized dogs and left in place for about an hour, at the end of which time the underlying tissues were deeply frozen. Frequent blood pressure, hemoglobin and rectal temperature readings were obtained. In all instances the blood pressure fell markedly before death and there was a marked blood concentration. In one experiment[13] the last hemoglobin reading was 168. At the close of the experiments the dead animals were carefully bisected by the author's method[6] and a comparison made of the weights of the frozen and unfrozen sides. The average difference amounted to about 34% of the total blood volume of the animals. Incision into the tissues that had been frozen revealed considerable plasma-like fluid, as verified by chemical analysis. It was concluded, therefore, that in these experiments the amount of plasma leakage was sufficient to account for part at least of the resultant shock.

Patients with frozen ears were observed frequently and in all instances after thawing a marked swelling was noted. Only one patient with extensive freezing injury was observed in this study. This patient, a woman, aged 43, tried to commit suicide by lying in the snow a night when the temperature was —22° F. When found she was not unconscious, but both legs were frozen stiff to just below the knees and when her stockings were removed they came off like wallpaper off a plastered wall. She received heat generally and cold compresses to the local lesions. The legs thawed out, but the toes of both feet and the sole of the left foot remained very syanotic. Later gangrene remained in the toes of the left foot.

One of the chief items of interest with regard to this patient was the extensive swelling and blistering of the frozen legs. The blister fluid was essentially similar to blood plasma on chemical analysis. Although the plasma loss in this case was not sufficient to produce shock, it indicates what might happen with a more extensive loss from freezing of a larger area.

General Changes in Body Temperature in Local Freezing: In the animal experiments previously described several very low rectal temperatures were recorded. In one dog a rectal temperature of 25.7° C. (78° F.) was noted. After removal of the dry ice, the temperature rose gradually to 33.7° C. (92° F.) at the time of death 16 hours later. Reincke[14] in 1875 reported a very interesting series from Hamburg of 17 drunken men exposed to extreme cold while intoxicated. On admission to the hospital all had markedly lowered rectal temperatures. Twelve of them did not fall below 30° C. (86° F.) and recovered. The other five had temperatures that went below that point, two of whom recovered. One patient had a temperature of 28.4° C., eight hours before death. Another had a temperature of 27° C. (80° F.) with death 13 hours later. The prize case in Reincke's series, however, was that of a 34-year-old laborer who was admitted with a temperature of 24° C. (75.2° F.). Four hours later the temperature was only 27.4° C.; 10 hours later only 32.6° C.; 23 hours later 37.8° C., and the day following he was discharged completely well.

It seems, therefore, that the effects of lowering of body temperature are to be reckoned with in cases of freezing.

Plasma leakage may be a factor, but it is not as important an item as in burns.

Light and Electricity

SUNBURN can occasionally be so severe as to cause death, presumably from shock. Extensive blistering over a large part of the body surface may be a morbid factor in this condition. Chemical burns may be similar to thermal injuries. Electrical burns produce shock in many instances by direct trauma to the central nervous system or by leading to ventricular fibrillation. Localized electrical burns produce deep tissue destruction. This comes on more rapidly than do roentgen ray burns. In general, however, the effects of light, electricity, roentgen rays, etc., do not produce the type of surgical shock produced by burns. The element of plasma loss is an even less important factor than in freezing.

Comment

CORRELATED laboratory experiments and clinical observations indicate that unrecognized blood loss is an important factor in traumatic shock. In severe burns shock is usually associated with a leakage of blood plasma-like fluid into the burned tissues and from the weeping burned surface. Tannic acid may be of value in decreasing this loss. Experiments indicate that leakage of plasma-like fluid in amounts hitherto unrealized is present in other conditions of which freezing is an example in kind. In the treatment of freezing the effects of rapid thawing and low general body temperature are

to be considered as well as plasma loss.

Actinic, chemical, electrical and roentgen ray burns because of the nature of their production seldom produce a type of shock similar to thermal burns although they could theoretically do so. It is to be remembered that the injury produced by all these external agents may result in a general condition of shock as well as the evident local trauma. The treatment of shock is often as important as that of the local lesion and is essentially similar in all instances.

Since the fluid that will be lost in these conditions is plasma rather than whole blood, the ideal therapy might be plasma infusion rather than whole blood transfusion. The writer has used plasma experimentally, but has not tried it clinically.

Conclusions

A SHOCK-LIKE condition is present following burns, freezing, and other similar types of trauma. The loss of plasma-like fluid into the injured tissues is a factor in the production of this shock. The treatment of the shock when present is of as much importance as that of the local condition. This treatment should especially emphasize restoration of the circulating blood volume by blood transfusion.

Low body temperatures following freezing are mentioned (78° F.).

Experiments to test the benefits of gradual warming up frozen extremities as opposed to rapid thawing showed no advantage to either method.

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THE BULLETIN

of

The American Association of Industrial Physicians and Surgeons

THE object of this Association shall be to foster the study and discussion of the problems peculiar to the practice of industrial medicine and surgery; to develop methods adapted to the conservation of health among workers in the industries; to promote a more general understanding of the purposes and results of the medical care of employees, and to unite into one organization members of the medical profession specializing in industrial medicine and surgery for the mutual advancement in the practice of their profession.

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The Cost of Fatigue

DR. H. B. TRUMPER, group labour manager of I.C.I. (Medals) Ltd., spoke on the cost of fatigue.* He said we talked of a man or woman being fatigued and, as a result, being more prone to accidents and sickness, not producing such good work and possibly causing dissatisfaction. No doubt all that was true, but merely to look at the net result did not take us very much further to the root cause.

Physiological experiments with a muscle nerve preparation had demonstrated that the muscle was the last part of the system to be fatigued; fatigue first occurred in the brain, then in the nerve, and lastly in the muscle itself. From these experiments it was seen that only extremely strenuous manual labour, as was very rarely done by men today, would produce fatigue, but fatigue did occur long before in the nervous system, and it was on account of this that it was so difficult to measure.

Attempts had been made to measure fatigue in various ways, but there was no scientific, no physiological, measure of fatigue. There was, however, a very important and very valuable measure—that of lowering of output. A reasonable definition of fatigue was: "a diminution of working capacity caused by length or intensity of some activity in a gainful occupation." Fatigue was an encroachment on normal reserve: we could see it in the faces of people

* At the Birmingham Conference, on October 7, 1936; Abstr. from *Labour Management*, November, 1936.

It was something indefinable, yet there to be recognized.

With regard to factors producing fatigue, on the physical side the most important factor was overtime, that is, working too long on the job. Secondly, there was abnormal time, that is, working at a time at which it is unnatural to work—night work, for example—and thirdly, undue exertion. The physical conditions which produced fatigue were exposure to harmful elements, bad or inadequate lighting, heating or ventilation.

On the mental side the factors were boredom, anxiety and insecurity; unfortunately all these three things were often present together. There were today many occupations of a repetitive nature where boredom was inevitable, and it was lucky that we had a section of the community who could withstand this fatigue, namely women.

With regard to insecurity, the average person did not appreciate the worry which came to the ordinary working man, especially if the man had the responsibility of a wife and family. When a man was constantly worrying about how long his job would last, and whether he would be successful in obtaining another, he was more likely to retreat into sickness.

Sick absenteeism could be divided into organic diseases and a huge group of indefinable disorders for which we had no real name: there were names which we could apply—psychological upsets—but there was no physical basis for them which could be demonstrated. Wherever you got a large class of indefinable absence, one fact stood out—fatigue must be present. There was a cure for it which was usually applied: fortunately in this country we could apply it—it was rest, and in most cases patients responded to it. When rest was applied, bodily functions were able to take on a new lease of life, as before they were overworked. This was as true of sickness of one or two days as it was of one week or so.

On comparing the sickness of office staff with that of workpeople, it was found that staff sickness was very much less, which was curious from the point of view that in most cases staff received their weekly or monthly wages whether they were away or not, whereas this was not so in the case of workpeople. Most staff men had one or two weeks' holiday a year, while many industrial workers did not, yet factory workers had as much as two weeks' illness a year. It might therefore be concluded that the workman in his sickness was making up what should be his rightful holiday. He was not ill but tired.

As a general rule it was assumed that fatigue was a possible or probable cause of accidents, for when men were doing hazardous work, keyed up all the time, their work might be so strenuous that they could only do it for short periods. The surprising thing was that these men were the safest to employ. They had very few accidents, whereas, on the other hand, in occupations not so hazardous, not so exciting, the accident rate was comparatively high. Tiredness and fatigue had not a great influence on accidents; there were other factors which we could not see at the moment, and the fatigue side was rather overstressed.

After an accident had occurred a man experienced unusual conditions; the atmosphere of the hospital was very different from that to which he had grown accustomed. As he recovered from his illness he enjoyed the period of convalescence, and got to look forward less and less to his return to work. He associated work with the hardness of the job, the noise of the machinery, the insecurity, temporarily forgotten, which he felt previously. He was likely to delay his return to work unnecessarily, and another cost of fatigue could be calculated as part of the accident absenteeism.

In Birmingham they were going through a peak period from the point

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of labour turnover. The city employed a large number of people on repetitive processes, with very little prospect at all; the immediate wage was the important factor. There was the extraordinary position of people who worked for one week, and changed for the sake of an extra shilling or so. He believed that large employers of labour would have been able to check this had they understood the social side, and endeavored to give their employees the sense of belonging to a community. The cost and lack of space were among the usual excuses for failing in this direction, but whether we could afford it or not, we were paying the price now, and he trusted that when the opportunity came we should get together and try to improve these conditions, so that when a person got into a good job, he did not think of a few extra pence a week as a reason to move to a different job.

No less than one-third of the cost of sickness and accidents could be attributed to fatigue. Taking the cost of sickness as 3% of the wage bill, one-third of that could be attributed to fatigue.

There were a large number of people who were carried by every firm. If one asked the departmental manager of almost any business concern how many people he considered were "carried" by other employees, he would probably say 1%; these were literally passengers in the department. They might be old servants of the company kept on for sentimental reasons, people who had sustained some injury from one type of work and had been transferred through pity or necessity to another department. The departmental manager put these "passengers" on what he considered light and unimportant work, e.g., cleaning windows, sweeping up the floor of the shop, etc., but it was just such jobs as these which made a difference in the lives of employees. In putting these men on the jobs mentioned, employers were making a double fault—they were putting the wrong men on important jobs, and they were carrying a passenger. He did not suggest that passengers should not be carried, but that it should be carefully decided in which compartment they should ride.

The conditions of work, that is, the privileges and wage a man received, were an important factor in methods of reducing fatigue.

In Birmingham it was unfortunate that we had district rates agreed with the trade organization for different trades. It was not possible to divide men up into various sections according to the work they did—fitters, welders, electricians, etc.—and suggest that every man in one group was worth the same amount per week. A man's wages should be decided by his value on the job; a man would appreciate the fairness of this, and if his job was important and carried with it responsibility, he should be paid accordingly. It was important that men should only be made to work reasonable hours: no man should feel that he got all the worst jobs — night work, abnormal time, etc. Each man should have an opportunity to fit himself for a better or more responsible job.

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Working environment included such things as heating and lighting. It was, of course, a good thing to order some windows for a department in the works where the employees would benefit by the improved conditions, but the value would be increased a thousand times if the people in the department had some opportunity of suggesting how the lighting, etc., should be arranged. Where changes were made and where improvements were put in, if the people concerned had an opportunity of commenting on them in the early stages, they would understand them and appreciate them.

Papilloma and Carcinoma of Bladder in Dye Workers

G. H. GEHRMANN points out that the first aniline tumors of the bladder to be reported in this country were detected in 1931*. Prior to 1914 European countries, principally Germany, manufactured about 80% of the entire output of dyes. It was subsequent to 1914 that the dye industry was developed on a large scale in this country. A period of 16 years elapsed before the first aniline tumors developed to the point of recognition in America. This period closely corresponds to the average time of exposure necessary to produce these tumors. Although it is accepted that certain chemical compounds of the nitro- and amino-group are responsible for the production of papillomas and carcinomas of the urinary bladder, the exact nature of these compounds is by no means clearly understood; neither is the mechanism of development. Experimental production of tumors of the bladder has been attempted for many years with predominantly unsuccessful results. The most important route of entrance is the respiratory tract, in the form of dust and fumes. Aniline is readily absorbed through the skin. The skin absorption of solid compounds such as alpha and beta naphthylamine is questionable. Absorption through the gastro-intestinal tract is probably of the least importance. However, it must be remembered that present knowledge indicates a long period of exposure to low concentrations as an etiologic factor. Therefore, any gastro-intestinal absorption should be considered as of more or less importance, despite the low solubility of these compounds. The age distribution in 24 cases of carcinoma shows a somewhat higher incidence after 30, but this no doubt is due to the insufficient time of exposure prior to that age. Five years appears to be the minimum and 25 years the maximum time of exposure for the development of tumors. The maximal period of exposure—25 years—represents the maximal period of exposure in the present series. More cases may develop as time goes on. These tumors continue to develop even after the patient is no longer exposed. In Germany they have occurred as long as 25 years after workers have changed their occupation. The classic symptoms of tumors of the urinary bladder are hematuria, frequency, urgency, burning and pain. These symptoms as a group occur in the author's experience only in those cases which are well advanced. The diagnosis in this series was made by periodic cystoscopic examination and the classification of the tumors by biopsy. Prior to cystoscopic examination, five of the 24 carcinomas showed hematuria. Symptoms of frequency, urgency and burning were so inconsistent as to be almost negligible as a diagnostic aid. It has been Dr. Gehrmann's experience that cystoscopic examination is the only safe method of early diagnosis of these tumors. Tumors may be single or multiple, papillary or sessile, infiltrating or non-infiltrating, ulcerating or non-ulcerating, malignant or benign. The histologic structure and treatment of aniline tumors does not differ from tumors of unknown etiology. Proper methods of plant control and medical supervision will eliminate their incidence. Exhaust ventilation designed to remove any dust or fumes that may escape from any of the equipment is essential. The final disposition of exhausted air is extremely important and the ventilation discharge should be sufficiently remote from all operations to prevent contamination in any area of the plant. Adequate measures of production in Germany have so successfully protected the workers that no new cases have developed in men who have been employed in these factories since the installation of their protective facilities. Every applicant for work in areas where he will be

* Abstract. In *Delaware State M. J.*, November, 1936, of DR. GEHRMANN'S article in *J. A.M.A.*, October 1, 1936.

OCCUPATIONAL DISEASES

The General Assembly of the State of Illinois has recently passed an Act, covering occupational diseases of Industrial employees. This Act is particularly concerned with diseases of the lungs, and poisoning from certain chemicals.

Under this new law it will be necessary for many industries to examine their present employees, and new employees, to determine their present health status and thereby prevent future liability.

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exposed to aniline, alpha and beta naphthylamine and benzidine should have a cystoscopic examination in addition to a regular complete physical examination. Any disease of the genito-urinary system is a contra-indication to employment, as is family history of cancer, history of hematuria or venereal disease. No applicant for this type of work should be accepted who is less than 21 or more than 40 years of age. All workers should have a complete physical examination and cystoscopic examination once a year.

Illinois Occupational Diseases Act

ON MARCH 16, 1936, Governor Horner signed a new Occupational Diseases Act for Illinois, which was passed by the Third Special Session of the Legislature.* This Act became effective October 1, 1936.

Prior to the passage of this new Act there was no remedy in Illinois for a man who became disabled due to an occupational disease unless the occupational disease was caused by some poisonous chemical, mineral or other poisonous substance. All non-poisonous dust diseases such as "silicosis" or "asbestosis" were not compensated for, even if the employer was grossly negligent in not using the proper preventive measures.

The new Occupational Diseases Act, by Section III, provides that where an employee in this state sustains an injury to his health or death by reason of a disease contracted in the course of his employment, and proximately caused by the negligence of his employer, he shall have a right of action against his employer for unlimited damages, except in cases of death not to exceed $10,000.

The so-called common law defenses are abolished. That is to say, the employer can not say that the disease which the employee received through the negligence of his employer was caused by a risk which the employee assumed in his employment, or that it was caused by a fellow servant, or that the disease was caused in whole or in part by the contributory negligence of the employee, where such contributory negligence was not wilful.

Any employer to whom this Act applies, and it applies to every employer of this state, except the farmer and private householder, may escape the stringent provisions of Section III by electing to provide and pay compensation in accordance with the compensation provisions of the new Act. If the employer so elects to provide and pay compensation, then the case is tried before the Industrial Commission and the question of whose fault it was that caused the occupational disease does not enter into the picture. If the employer elects, the Act defines an occupational disease as meaning a disease arising out of and in the course of the employment. Ordinary diseases of life to which the general public is exposed outside of employment are not compensable, except when the disease follows as an incident of an occupational disease as defined in the Act.

The employee, in case of disablement from an occupational disease, is entitled to all necessary medical, surgical and hospital treatment which will reasonably effect a cure, except in cases of "silicosis" or "asbestosis," because these two diseases are recognized as incurable. The liability of the employer is limited to a period not to exceed six months in this case.

Practically 23 of the 34 sections which compose the new Occupational Diseases Act were taken from the Workmen's Compensation Act, which takes care of accidental injuries. Under the new Act it is the duty of the employee to notify his employer as soon as practical after disablement from an occupational disease. A demand for compensation must be made within six months after the occurrence of disability from occupational disease, and an application for compensation must be filed with the Industrial Commission within one year after the date of the disablement, or within one year after the date of the payment of the last compensation, if any has been paid.

The employer who is liable under the provisions of this Act is the one that last exposes the employee to the hazards of an occupational disease, and an employee is conclusively deemed to have been exposed to the hazards of an occupational disease when for any length of time, however short, he is employed in an occupation or process in which the hazard of the disease exists. In case of "silicosis" or "asbestosis" the only employer liable is the last employer in whose employment the employee was last employed during a period of 60 or more days after the effective date of this Act. The purpose of that last provision was to make certain which employer was liable. It may easily be that an employee may not have been exposed long enough in an employment to receive the full effect of the disease, but to make certain whom he shall sue it was deemed best to make the last one that exposed him to the hazards of the occupational disease liable.

Like the Workmen's Compensation Act, it is the duty of the employer to furnish the necessary medical treatment, but the employee may choose his own doctor at his own expense. The employer has a right to reasonable examinations after disablement to determine the condition of the man and to prepare a defense in case he is sued under the compensation features of this Act.

Whenever, after the death of an employee, any party in interest files an application for adjustment of claim under this Act, and it appears that an autopsy may disclose material evidence as to whether or not such death was due to the inhalation of silica or asbestos dust, the Industrial Commission, upon petition of either party, may order an autopsy at the expense of the party requesting same, and if such autopsy is so ordered, the commission shall designate a com-

* PHILIP H. KREUSCHER, M.D., F.A.C.S., Medical Director, Illinois Industrial Commission; Editorial in *Illinois M. J.*, December, 1936.

The Association for the Advancement of Industrial Medicine and Surgery

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petent pathologist to perform the same, and shall give the parties in interest such reasonable notice of the time and place thereof as will afford a reasonable opportunity to witness such autopsy in person or by a representative.

It shall be the duty of such pathologist to perform such autopsy as, in his best judgment, is required to ascertain the cause of death. Such pathologist shall make a complete written report of all his findings to the Industrial Commission (including laboratory results described as such, if any). The said report of the pathologist shall contain his findings on postmortem examination and said report shall not contain any conclusion of the said pathologist based upon the findings so reported.

By that it is meant that the pathologist is to give only his findings, and not conclude that the cause of death was, for instance, arsenical poisoning. In other words, he may give all his findings, which may go to make up his conclusion of arsenical poisoning, but his conclusion as to the cause of death must not be contained in the report.

The report of the pathologist is filed with the Industrial Commission, and becomes a public record. Either party may produce any evidence to rebut any part of the report.

If an autopsy is performed at any time without the express or implied consent of an interested party, when the opposing party is known or reasonably ascertainable, and without the said interested party being given reasonable notice and reasonable opportunity of witnessing the same, all evidence obtained by such autopsy shall be barred upon objection at any hearing. This does not apply, however, to autopsies by a coroner's physician in the discharge of his official duties.

Under the compensation features of the new Act, a man is paid for disability which is defined as the event of being disabled from earning full wages at the work in which the employee was engaged when last exposed to the hazards of the occupational disease by the employer from whom he claims compensation or equal wages in other suitable employment.

No compensation is payable on account of an occupational disease unless disability as defined in the Act occurs within one year after the last day of the last exposure to the hazards of the disease, except in case of occupational diseases caused by the inhalation of silica or asbestos dust and, in such cases, within three years after the last day of the last exposure to the hazards of such disease.

During the regular 1935 session of the Legislature, attempts were made to pass various acts relating to occupational diseases and they failed. A companion measure, known as the Health and Safety Act, gives the Industrial Commission, after a hearing, the right to make reasonable rules which will have the force and effect of law for the prevention of personal injuries and diseases. In carrying out the details of this Act, consultation and advice of physicians will be necessary and essential. The medical provision will play an important part in carrying out the duties imposed by the new laws. The main objects of these laws are, first, prevention; second, adequate care; and third, reasonable compensation.

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Common Occupational Diseases

ABOUT 900 industries are recorded under the heading of hazardous occupations.* As time and patience are limited, let us discuss a few of the problems in broad, general terms, rather than in minute detail.

Probably the leader in the parade of industrial diseases is an acute or chronic inflammation of the skin which we call dermatitis. Rare, indeed, is the industry which has not placed such a case upon its lost time list, perhaps for days or perhaps for years. This disease may present itself in any type of shop, factory or foundry. Any paste, powder, gas or liquid which comes in contact with the employee's skin, even the water supplied by the town in which he lives or works, may be the potential cause of such a skin disturbance. Even under this very roof, the chambermaid, vegetable cook or the baker may become afflicted with dermatitis, the former from irritation of the soaps and bleaches of the clean linen; the cook from insecticides on the vegetable tops which she prepares for cooking and the baker from the flour which is to be made into bread. The wide diversity of cause together with the varying susceptibility of different individuals makes the problem of control of this disease most difficult. It causes much lost time from work and much discomfort to the patient.

It may be interesting to note that much investigation is under way to determine, if possible, why one per-

* Dr. Robert B. Hunt, Medical Adviser, American Mutual Liability Insurance Company; Presented at the Occupational Diseases Section, 25th National Safety Congress, Atlantic City, N. J.; published in *American Mutual Magazine*, December, 1936.

DUST IN INDUSTRY — What the hazards are; how to determine their existence; how to combat them.

INDUSTRIAL DUST

Hygienic Significance, Measurement and Control

By Philip Drinker *and* Theodore Hatch
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316 pages, 6x9, 104 illustrations,
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This book covers the problem of dust control in its relation to the health of workmen, emphasizing the cooperative nature of the problem both for physicians and engineers, and presenting principles, methods and data for the design and operation of dust-control equipment. It discusses the physical aspects of dust and fume suspensions and their effect upon man; the analysis, measurement, and microscopy of fine dusts; and the practical control of dusts and use of respiratory protective devices.

INDUSTRIAL MEDICINE
844 Rush Street,
Chicago, Illinois.

son may be susceptible to a certain substance while another is not. It has been proved many times that a poisonous substance formed within the body has been able to prolong the action started by some external influence, and I should like to tell you about two such cases.

A printer developed a marked dermatitis from ink which persisted for two years. Finally, food tests were made and showed that he was susceptible to lettuce, which, when removed from his diet cleared the skin. He returned to his usual occupation without a return of symptoms. Recently, in a large electrical shop, the men were re-equipped with new, heavy rubber gloves. Three of these men developed a dermatitis of the arms and hands. Why were three out of the 120 men so affected? These three men were subjected to food tests and each reacted to a specific article of diet. When that offending element was avoided, the skin lesions cleared, the men returned to their usual occupation, again put on the offending gloves, and had no further trouble.

The poisoning of allergen substances is usually an accumulative process. It takes time to produce a manifestation of disease but let any other substance cause the disease and the irritating food may be of sufficient strength to magnify its action. It is much like pushing an automobile—it requires a great deal of strength to start it but little exertion to keep it moving.

I do not contend that all cases of dermatitis can be cured by eliminating offending foods but I do think that further investigation along the same or similar lines might give us a cue with which to lessen the loss of time from work and the discomfort which is now produced by industrial dermatitis.

Quite diversified are the uses of lead and equally extensive is the distribution of lead poisoning.

That you may have a better understanding of the body's reaction to this metal and hence a better insight into the hazard of the employee's occupation, let me state briefly a few medical facts.

The skin of the body is a protective covering and as long as it remains intact does a very good job except in the case of tetraethyl lead which is used in blending gasoline and which is readily absorbed through the skin, having a tendency to create changes in the nervous system. The effect of other forms of lead, through that avenue of entrance, is practically nil, which explains the extremely low poisoning rate in typesetters, painters and others who continually come in contact with metallic lead.

Lead, in any form, introduced through the mouth acts rather slowly when compared with the inhalation of lead dust or fumes. However, no matter what the portal of entry may be, lead is acted upon by the body fluids and so converted that it may eventually be deposited within the bones.

Those bony safe deposit vaults may retain their lead contents for days or for years, and if the exposure is removed, may also free the employee of all signs and symptoms of lead poisoning. But imagine the surprise of that supposedly cured person when years later, perhaps following some type of infectious disease or the excessive use of alcoholic beverages, he awakes to find that he suffers from the aches and pains and paralyses of an acute lead poisoning. As the body fluids alter lead that it may be deposited, so may those fluids attempt to delead the body and too rapid absorption may produce the original disease with all its discomforts.

One can readily see, then, that in some cases investigation of previous occupations is quite necessary to determine the original contact with lead.

Heating of molten lead causes a black scum of lead suboxide to form on its surface and the heat from the metal disburses the fine powder into the air. Hence, in the process of smelting ores such as zinc, copper, silver, etc., mixed with lead, we find a rather high lead poisoning rate.

Those engaged in the manufacture of lead wire and cables, batteries, in glass, pottery and rubber industries, plumbers and users of dry colors offer their share of lead poisoning.

Probably the greatest source of lead poisoning may be found among those who sandpaper, scrape, chip or burn lead paint, for the dust and fumes caused by those activities are rather rapid in their action. Such workers may become incapacitated in a few days because the inhalation of lead dust causes the maximum amount of absorption into the blood stream in the minimum space of time.

Although this risk is outside the field of hazardous occupations, an employer or employee may find lead poisoning in his offspring as the result of gnawing and eating the enamel off the crib in which he sleeps.

Chromium is a metal which is gaining in popularity and with its advance in usage comes the consequent increase in its effect upon man. It is a widely used foundation for colors, and dusts from its various salts may produce lesions of the skin and mucous membrances of the nose. Chromium plating by electrolysis may cause fumes which are similarly irritating. Although chromium poisoning does not usually cause death, the number of days of absence from work on account of incapacity which it causes is gradually increasing.

The Industrial Doctor

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The most efficient portal of entry of foreign substances into the body is through the nose and mouth. The smaller the dust (the size of smoke particles for example) the greater the speed of absorption by the blood. Fumes from hot metals carry not only poisons but numerous minute dust particles. Such facts explain the incapacities occurring among those engaged in acetylene and electric cutting and welding. When men are so employed over a prolonged period of time, the constant inhalation of fumes and dust might produce symptoms of the metal upon which they work. In the ship-building trade, particularly in the construction of metal ships, we find occasional cases of metal-fume fever. "Brass chills" and "spelter shakes" from zinc are names applied by the lay people to such diseases. The terrific heat applied to some of our common metals and alloys by acetylene torches produces sufficient fumes to cause a temporary incapacity in the short space of 24 hours. Permanent disability from such causes is rare, however.

Twenty-five per cent of all workers exposed to high concentrations of inorganic dust are expected to develop changes within their lungs due to that agency. We are at a loss to explain why the other 75% are not so affected.

Such occupations as mining, quarrying, foundry work, grinding, sandblasting, abrasive soap manufacturing, asbestos work, the making of emery and carborundum products and many others offer opportunities for employees to inhale microscopic dust particles and create a foundation for possible future incapacity.

We are interested particularly with those men who work with sandstone, quartz, flint, granite, and sand because they expose themselves to silica and the chemical changes between that dust and the body fluids, in the presence of tuberculosis may and usually does lead to a fatal ending.

I think it is safe to say, in light of our present knowledge, that the action of other inorganic dusts is much less virulent and incapacitating. That statement, however, does not allow us to lower the vigilance of methods to improve dust control and protect the employee because all dusty occupations are potential trouble makers.

Although the actions of various dusts differ within the body, the exposure to asbestos fibers, present in the weaving and grinding of dry asbestos materials, offers another type of dust which may cause fatalities among workers.

The organic dusts from cotton, silk, wool, tobacco, flour, etc., are practically harmless. Such dusts rarely reach the lungs but may cause irritation of the windpipe and larger air tubes and result at the most in a mild bronchitis.

The factor which should be borne in mind, particularly when dealing with silicosis and asbestosis, relates to the progression of such disease. When changes occur within the lungs as a result of the presence of such dusts, nature forms scar tissue to heal such lesions but may fail to stop when the job is finished. As a result, the continuation of that healing process may lead to such structural changes within the lungs that partial or total incapacity may follow months or years after the employee has been removed from his dusty occupation.

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Workmen's Compensation

ATTENTION has been called to the racket, participated in by some, of being subpoenaed regularly on all cases, and warning has been issued that only when the carrier, employer, referee, Industrial Commissioner or Council serves the subpoena will such attendance be paid for.* Too often an attempt at misrepresentation is made—the subpoena really being served by someone in the employ of the claimant, the employee.

Another racket is coming to the surface. Employees are learning that physicians have different opinions as to the degree of disability and the length of time before they can return to work. As such information is spread about, some employees are choosing physicians who will allow them the greatest degree of disability and the longest time off.

These various rackets which we cite from time to time are venomous and it would be well not to have your name connected with any of them. The medical profession has been made responsible for the operation of the Workmen's Compensation Law at its own request. Our chief concern is the best interest of the workman. He can best be served by honest methods—conniving and cheating in any way only reflect discredit upon the physician, and do harm to the employee. It is not only the right thing to get the injured workman back on his job as early as possible, but it is most helpful to labor generally and to the individual employee particularly. It maintains his self-respect to be returned to the job of providing for his family needs as soon as possible, not just receiving the small weekly stipend of compensation; it sets a good example to his growing sons and daughters and to his fellow workmen; it takes from him any resentment that he may hold against one who may have been the cause of his accident; it allays fear as to the possible outcome of the injury on his health and well being. If a long convalescent period intervenes, the employee is obsessed by the fear of future economic insecurity. These

* From *Bulletin of the Medical Society of the County of Kings*, December, 1936.

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conflicting thoughts naturally delay the settlement of claims, not overlooking the fact that, in the case of the average individual, the delay adds to the worry over the uncertainty of the final legal status of his case and so prolongs recovery. It is a vicious circle, and the physician who makes every effort to return his patient to work, even to light work, as early as possible, is serving the public more than he may appreciate.

Dr. David J. Kaliski, Chairman of the Committee of Workmen's Compensation of the Medical Society of the State of New York, has called attention,[1] to the need of all physicians reporting all cases of compensation accidents to the Department of Labor and not heeding requests of some employers to treat their employees without such reports. Let us not violate the law.

Illinois Advisory Committee

THOMAS C. ANGERSTEIN, in his article elsewhere in this issue, notes that the Illinois Industrial Commission, under the recent Health, Safety and Comfort Act, has the duty and power to appoint an Advisory Committee.

Peter J. Angsten, Chairman of the Illinois Industrial Commission, has appointed the following individuals to represent the employer and the employee as members of such Advisory Committee:

Employer representatives:

O. E. Mount, Secretary, American Steel Foundries; J. L. Earlywine, Attorney, Carnegie - Illinois Steel Company; J. H. Chivers, M.D., Medical Director, Crane Company; H. G. Hensel, Safety Director, Youngstown Sheet & Tube Co.; E. Bornstein, Secretary, Caterpillar Tractor Company, Peoria.

J. W. Freels, Attorney, Illinois Central Railroad.

(One additional representative will be appointed.)

Representatives of organized lobar:

R. G. Soderstrom, President, Illinois State Federation of Labor, member, Typographical Union of Streator.

Jacob Benneman, member, Legislative Committee, Chicago Federation of Labor, and Engineers' Local Union.

George Ottens, representative, State Carpenters' Council.

Samuel Levin, Manager, Chicago Joint Board, Amalgamated Clothing Workers.

J. J. Uhlmann, Secretary, Metal Trades Council.

Robert G. Fitchie, Vice-President, Illinois State Federation of Labor; President, Milk Wagon Drivers' Union.

Victor A. Olander, Secretary-Treasurer, Illinois State Federation of Labor.

Mr. Angsten will act as Chairman of the Joint Committee.

The Illinois Industrial Commission has the responsibility under the new Health, Comfort and Safety Act to promulgate rules and regulations pertaining to health, comfort and safety in Illinois industry. Their rules and regulations will, for all practical purposes, have the force and effect of law. This Advisory Committee will make recommendations regarding such rules and regu-

1. In the October 15 issue of *New York State J. Med.*, p. 1571.

SUPPLEMENTARY INFORMATION

A POSTCARD addressed to Industrial Medicine with your name, and the number as below, or
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lations to the Commission, which in turn will hold public hearings before rules and regulations are finally promulgated.

The members representing industry on this Advisory Committee are also members of the recently appointed Workmen's Compensation-Health and Safety Committee of the Illinois Manufacturers' Association — which consists of 48 members, of whom 44 are executives of industrial concerns belonging to the Association, two are industrial physicians and surgeons, and two are from the legal profession. Oliver E. Mount, American Steel Foundries, is Chairman, and Clark D. Bridges, newly appointed Director of the Workmen's Compensation-Health and Safety Bureau of the Association, is Secretary of the Committee. Dr. Joseph H. Chivers, Medical Director of Crane Company, Chicago, and Dr. Roy J. DeMotte, Chief Surgeon of the Pullman Standard Car Manufacturing Company since 1923, are the medical members; Edward A. Zimmerman, of Zane, Morse, Zimmerman and Norman, and Joseph L. Earlywine, of counsel for Carnegie-Illinois Steel Corporation, are the lawyer members.

Industrial Nursing

THIS is a brief resume of the medical work as carried out in the Myer Emporium, Melbourne, Australia.*

Any member of the staff is eligible to ask advice at the Medical Department. The nurses see the patients first, drafting off those whom we think need more expert investigation than we can give them, to the doctor. This is done as far as possible by appointment, but of course there are always the urgent ones with whom to be dealt.

Many are under the care of the private practitioner and when off work must call in a doctor or go to the hospital if funds allow. On return to work they must report to the Medical Department to be certified fit for duty. Accident cases away for a week or more come under the Workers' Compensation, which necessitates the filling in of particulars as to time and place, nature of accident and probable duration.

Many wonder just what a doctor and two nurses can find to do in a store where everyone looks comparatively well. They will suggest that we are sure to have a few faints. This is just an outline of what we do have:

Heart and arteries: High blood pressure; rheumatic heart, congenital heart disease. These are cases not sufficiently severe to prevent working.

Lungs: Tuberculosis; pneumonia; pleural effusion; blood-stained expectoration. These are referred to their own doctor. They may be lodge or hospital patients.

Nervous conditions: From cerebral tumor to anxiety neurosis often due to stress and strain of modern business.

Blood: All degrees of anemia.

Miscellaneous: Every variety of rheumatism, diabetes, duodenal ulcer, gastro-enteritis, fractures, joint injuries, hematomia, infected hands, burns, boils, carbuncles. Acute appendicitis crops up every now and then, and we had one case of volvulus.

In the midst of these things there will be a call to see a customer who has taken a bad turn in the shop. The assistant who rings the Medical Department probably does not give much idea of what the trouble is—just that the lady looks sick. On reaching the patient the nurse, who was taught in hospital not to diagnose, must quickly make up her mind to do something. These excitements have a habit of happening in the afternoon when the doctor is not available.

Should the doctor find that one of the junior members is under-nourished or in poor circumstances or obviously in need of a holiday, her home life is investigated, and in conjunction with the staff office an arrangement is made for her to have a holiday in the country or a rest home. Members of the staff contribute voluntarily towards maintaining a fund for such things. Twenty-seven such cases were sent through the year to Healesville (a rest home).

* Olive McLeish, in *Public Health Nursing*, December, 1936; condensed and reprinted in part from *Una*, the Journal of the Royal Victorian College of Nursing, Melbourne, Austalia, January 1, 1936.

Permissible Dust Concentrations

THE Advisory Committee on Dust Control Rules and Regulations for Rock Drilling Operations, submitting rules and regulations to be recommended to the New York State Industrial Board for inclusion in the State's Industrial Code, propounds definitions:

"Silica-bearing rock" means a rock formation having as a component part any free silicon dioxide.

"Rock drilling operations" shall mean drilling, cutting, chipping, channelling, broaching or crushing to excavate rock by means of machinery or hand hammers.

"Injurious silica dust concentrations" shall mean dust produced from drilling silica-bearing rock which is in excess of 10 million particles per cubic foot as determined by an approved dust count method.

In determining the definition of "injurious silica dust concentration" as more than 10 million particles per cubic foot, the Committee acted on the advice of a Medical Sub-committee which submits a "Memorandum on the Subject of Permissible Concentrations: There are two avenues of approach to the problem of setting a permissible limit of dust concentration in rock drilling or any other dusty industry. One is based purely on the medical evidence as to the level of dustiness below which pulmonary damage cannot ordinarily ensue. The other is based on a level of dustiness which can be readily attained by engineering control by means of devices now in practical use and which are not excessive in cost.

"The Medical Sub-committee originally approached this problem from the first viewpoint and at the meeting on October 8 presented a sliding scale of dustiness wherein the allowable dust concentration varied inversely with the percentage of silica present in the dust. Without question this tabulation is justified by the best medical evidence available, and recognizes the well established fact that all dusts are not equally harmful. For this reason, it permits higher allowable concentrations in dust of low quartz content and lower permissible concentrations of dusts high in quartz content.

"From the viewpoint of the practical administration of such a code, there are however, serious objections to such a sliding scale: (1) the amount of inspection work would be greatly increased and the nature of the inspection would require a technical knowledge that cannot be provided without special training; (2 the frequency of sampling and the volume of laboratory work involved would be beyond the capacity of the staff to carry out; (3) it would offer unlimited opportunity for controversy over the question of compliance, especially in the case of a rock whose composition is subject to rapid change as the work progresses, e. g., when quartz veins are encountered in a rock normally low in silica; (4) the sliding scale would confuse the problem of testing dust control equipment for approval. Since such equipment will be used on all kinds of rock, it is clear that every piece of apparatus must pass the most rigid test. There can be no sliding scale of approval but if the code recognizes different levels of permissible dustiness, it will be difficult to explain why a piece of equipment which fails to give adequate control against quartz-rich dust should not be approved for use against dust containing a small percentage of free silica.

"It seems obvious that the adoption of such a sliding scale would set up code requirements which could not be enforced and our code, therefore, would be worse than useless. We would not cope with the problem at hand but would defeat the ends which we are all so deeply interested in attaining.

"The engineering approach to the solution of this problem is an approach based on the general requirement that engineering control methods shall be instituted, which methods represent present-day, good engineering practice readily attainable by means not prohibitive in cost. This approach was promulgated over 20 years ago by the Massachusetts State Department of Labor and has been in use in all States of the union without most of us being conscious of it. For example, in all industrial codes pertaining to grinding and polishing, foundry practice and all other types of mechanical and health protection we have been guided by this basically sound principle. In this connection, it is interesting to note, that no matter what medical standards are arrived at if such standards cannot be attained by engineering techniques now available, they cannot be promulgated because industry would not be in a position to comply with them. The New York State Department of Labor takes the attitude that industry should not be asked to reach standards of any kind which cannot be attained by good engineering techniques available at this time.

"A consideration of the engineering techniques available for the protection of the rock driller against the dust hazard in his occupation leads us to believe that a level of dustiness of five million particles or

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less can be attained in such operations by engineering techniques which are now available at a cost which is not prohibitive and based on an engineering approach which is practical and which may be attained by reasonable means at a cost which is in no way prohibitive. We believe that this figure should constitute a reasonable standard for this industry."

Syphilis Among Workers

IN AN industry employing more than 36,800 employees, with plants in 17 different states, the control of syphilis has presented special problems to the Medical Director.* Since 1934 the medical service of this company has tested all new employees and as many of the old employees as would voluntarily submit to a serological test for syphilis. Of 36,794 employees examined, 4.2% were found to be positive for syphilis through a serological test. These figures indicate that the Medical Director of this company and his superiors are fully aware of the implications in industry of syphilis, and certainly warrant commendation for the expense to which they have gone in setting up this special adjunct to an already-existing wel-established medical service. It will be wise for a large number of industrial establishments to do likewise, as the full implications of symphilitic infection are often most serious. Experience in the plants of this large corporation indicated that very few of the workers found with postive serological test refused to take treatment; many of the patients were being treated inadequately due to lack of adequate income or due to the current high fees for medical service in their own communities.

* *Safety Engineering*, November, 1936.

Dermatitis

SOME common errors in the determination of the industrial nature of a dermatitis.* The dermatitis is erroneously considered to be industrial because:

A. It is a dermatitis venenata occurring in a worker in an industry in which dermatitis venenata is known to occur. (For example, such a dermatitis venenata may be due, in the specific case, to non-industrial exposures—home exposures to dyes, cosmetics, toilet articles, insecticides, clothing, paint, varnishes, plants, etc. —exposures to substances encountered in hobbies or avocations—photography, painting, sports, etc.)

B. It is accompanied by a positive patch test of a substance or substances encountered in the industry. (Such a positive test cannot be considered conclusive, for it may be due to: (1) the application of a primary irritant or of a primary irritant concentration; or (2) the fact that the test never accurately reproduces the conditions of actual industrial exposure; therefore, a positive reaction may mean simply a certain skin sensitivity to a certain substance when applied in a certain concentration and manner at a certain time to a certain site, while this same substance may nevertheless be incapable of causing dermatitis under the actual conditions of industrial exposure.)

News

PAUL A. DAVIS, M.D., Assistant Medical Director, Goodyear Tire & Rubber Co., Akron, Ohio, Representative of the Section on Preventive and Industrial Medicine and Public Health of the Scientific Assembly of the American Medical Association, has been appointed to the Scientific Exhibit for the Atlantic City Session.

THE chemistry and physics of glass surfaces are being investigated at Mellon Institute of Industrial Research, under the auspices of Bausch & Lomb Optical Company, of Rochester, N. Y., with Frank L. Jones, Ph.D., a member of the staff since 1931, in charge. The investigation, to aid in the development of scientific apparatus and ophthalmic instruments, will be concerned with the effects of environmental factors on the durability of the glass used in optical instruments.

UNITED States Commissioner Cyrus W. Phillips, 66, known as "the Father of the Workmen's Compensation Law," died at Rochester, N. Y., December 2. In 1910 he drafted the first Workmen's Compensation bill in the United States.

REPRESENTATIVES of the Consolidated Edison Company, the New York State Department of Labor, and manufacturers of hat blocking machinery conferred (December 7) on the problem of carbon monoxide poisoning in the hat industry during the winter months; decided to launch a series of practical tests of new types of gas burner for hat machinery.

* MARION B. SULZBERGER, M.D., *New York State J. M.*; Reprinted in *Ohio State M. J.*, December, 1936.

Reproduction of an actual field showing dust particles and counting scale.

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1. Holmes, Pigott, Sawyer & Comstock—Indust. Med., July 1936.
2. Beard—J. Am. Dietet. A.—10:193 (1934).
3. Gardner & Gardner —Am. J. Dis. Child. 47:1261 (1934).
4. Cameron — J. Am. Dietet. A. 11:189 (1935).

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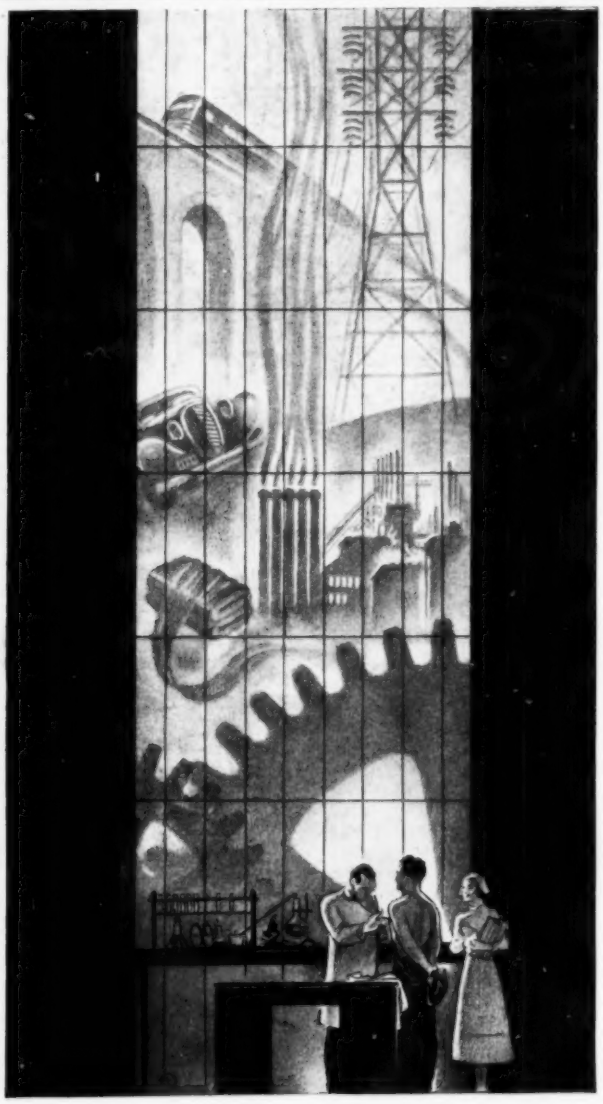

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